



**THE MANAGEMENT OF CANCER IN TOTALITY
INDIA CAN TAKE A LEAD IN THEORY AND APPLICATION**

Vol - 1

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VOLUME 1



**INDIA CAN TAKE A LEAD
IN THEORY AND APPLICATION**

Diagnosis ■ Treatment ■ Rehabilitation ■ Terminality

Dr. ASIM CHATTERJEE

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The Management Of Cancer In Totality

by Dr. Asim Chatterjee

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on behalf of Dr. Asim Chatterjee, Author.

Dedicated to
my brother-in-law Late Pandit Surendra Nath Chakravorty
(Vedashastrî) & my elder sister Late Ira Chakravorty



*For
my father
Late Rabindra Nath Chatterjee*



FOREWORD

Cancer, the very name, creates awe in everybody. It reminds of lethal stings of a crab. It's like a hunchback demon in our body, an octopus in life, and a disaster in today's nuclear families. Its backlash on health, humanity and economy is enormous.

Not that it is new to us; nor even that it is an alien in our system. Hundreds and hundreds of years ago, it had been spotted by the Oriental Medical System or Vedic Medical System known as Ayurveda which used to be known as the Fifth Veda. In Ayurveda, it used to be known as "Arvud" meaning lump. Shushruta identified cancer of two types – Adhyarvud and Dwirarvud (Shushruta 2.11.15). Charaksamahita also spoke of cancer, and recommended or the use of "Upodika" (botanical name: *Basella alba* *din* and *Basella rubra* *din*) as a cure. We get similar references in another very ancient compilation of Chakradutta. Charaksamahita as well as Shushrutasamahita often mentioned about the use of Kadamba (botanical name: *Adina cordifolia*) and other indigenous herbs as a cure or gland cancer, as long back as in 1st A.D. Ayurveda thus identified the prevalence of various types of cancer. "Shalya-chikitsa" or surgery also used to have formed a part of the Ayurvedic system of treatment, but exact details of the same are perhaps lost in course of time due to growing popularity of western medical systems of curing diseased actions by administering opposite actions including preventive actions. Nobody claims that such cure as prescribed in Ayurveda was tested as fool-proof or sure enough, besides the methodology and the procedure for preparation as well as use of such medicines having been too complicated. Over the time, the Ayurvedic cure started losing its ground, while the Allopathic system of treatment emerged as the best alternative as a scientifically proven cure to a large extent. Similarly, Homoeopathic system of treatment is out and out symptomatic and lacks in any in-built diagnostic procedure of its own, unless combined with the Allopathic procedures of various pathological and radiological tests. The Allopathic treatment of cancer normally consists of pre-treatment diagnostic procedure, post-diagnosis treatment associated with surgery, and post-surgery therapies like chemotherapy, radiotherapy etc. the whole system is not only a very costly affair, but also sometimes unbearable, particularly when such patient is at the terminal stage. Yogic therapists term cancer as infected fibroid which affects lymphatic vessels of the body and according to them, existence of too much to toxic substances in the body due to harmful hod-habit, imperfect life-style etc. are the root cause of cancer. They prescribe various yoga exercises, typical food, disciplined life-style and numerous breathing exercises on daily basis,

beyond the capacity of most of such patients. Moreover, such therapies discourage taking of any medicine, while it is not possible for a cancer patient to resort to such rigorous practices in the ailing health condition.

Therefore, there has been a long search for a low-cost, less-painful, hassles-free, and comprehensive therapy which can render a nearly-full sigh of relief to a cancer patient as well as to his or her family. It's not easy, everybody knows. Thousands of doctors and scientists are on the hunt for a really effective single-line solution for the disease. In this journey for light and life to cancer-victims, no one is trivial or insignificant. It is often said, an ancient Greek mused about the meaning of life, and Philosophy was born; the first Roman resolved to build a road instead of cutting a path through a jungle, and engineering came into being; one day in primitive times, somebody lent to some other whatever then passed for money and got back his original investment plus a little more, and banking started. None of them was known to have been academic scholars or educationally tall. Many of the landmark inventions and trend-setting innovations in the world came from persons having vision and zeal for doing something good for mankind. As we know, many scientific achievements have come from persons simply rich with a rare scientific mind and acumen. Mission has to be set firm, vision has to be kept clear, while efforts have to be sincere and honest with an ignited mind. To combat cancer, it's not the only need for invention of unfailing medicine, but for the invention and successful innovation of a complete or comprehensive therapy which may consist of pre-diagnostic procedure economically accessible to all, less-complicated treatment procedure after detection, and less-complicated post-treatment procedures. All these procedures need again be dovetailed with counselling at each stage. In this search for a comprehensive therapy for cancer-patients, Dr. Asim Chatterjee and his son, Dr. Aradeep Chatterjee, have undoubtedly made an indelible mark. I have had dozens of sittings with Dr. Asim Chatterjee on the subject of devastating effect of cancer on human life, on the affected families, society and its economy, and on the whole mankind. We have had rounds of discussions on the totality of cancer-treatment and the need for prior counselling as well as post-recovery counselling not only to a cancer-patient but also to his or her family-members who often pose to be the resultant victims. According to Dr. Chatterjee, cancer victimizes not only the patient but also his or her entire family and leaves a perennial impact on their future, for which he advocates for appropriate rehabilitation policies particularly for marginal families even from the Government or big corporate Houses' side, in case the sole bread-earner succumbs to the deadly claws of cancer. He is very serious in his approach when cancer is talked about.

Incidentally, more than 20 years of my service-career in many important positions in the Government have been spent in such a tribal area where cancer's killer claws are well-felt. A whole population so far having stout physique is slowly and steadily being the victim of cancer. In my capacity as the President of Rotary Club of Aizawl as well as Member of various District Committees of the Rotary International, I used to voice my concern about cancer in many seminars and was instrumental in holding Cancer Awareness Camps on a number of occasions. Within the Government, I often ocussed on the urgent need for organic farming to reduce the perils of pesticides used in common farming mode and the like. In fact, when no other State in India made any law on organic farming, on my advice and as drafted by me, Mizoram enacted the Mizoram Organic Farming Act, 2004, as a measure to combat cancer.

Dr. Chatterjee undertook a long journey in search of 'Vishalyakarani' for the cancer patients on his own without any Government aid or any financial or logistical assistance from any corner. His journey was not smooth at all. The road chosen by him was full of potholes, ditches, blind turns, and at the same time narrow. Still he did not fall like mythological Arjun before the end-point. He carried himself even in the darkness in the light of ample knowledge and able guidance of his erudite well-wishers about whom he has shown his gratitude in his book again and again. His Psorinum Therapy for treatment of cancer is now well-recognised not only in India, but also in many other developed countries. This book introduces the Psorinum Therapy and its background details including Dr. Chatterjee's personal views on totality of cancer treatment to the people. It speaks about his research days, test-results of his therapy on many terminal patients of renowned hospitals or medical institutes, setting-up of his own cancer research and facility-centre for cancer treatment, and many unknown events of his journey towards an effective cancer cure. We all hope that his efforts must not die only with his therapy. His journey must not end here. More doors he may be able to open for cancer-patients, I sincerely believe.

I wish the book a great success. I wish Dr. Chatterjee more feathers of many more colours in his cap.

Prithwipati Chakraborty

M.A (Econ.) LL.B. etc.

Ex-Principal Secretary, DCA Deptt.

Principal Legal Advisor, Govt. of Mizoram

Judge, Appellate Tribunals, ADA & Aizawl

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Mr. Prithwipati Chakraborty

PROLEGOMENON

Dr. Jaydeep Biswas

MBBS, MS, FICS, DMRT, FAIS, Double FRCS.

Director of Chittaranjan National Cancer Institute, Kolkata

HoD, Surgical Oncology

At the outset it should be mentioned that Dr. Chatterjee's research work on cancer is indeed commendable and is a mark of bravery. For which I have deep respect for him. the most notable thing is that he has always followed the scientific way though he has not come to the cancer research field from the conventional background. He is neither against conventional cancer treatment nor has he ever passed any adverse remarks to any patient regarding conventional ways. I always felt inspired and remain attached towards his honest effort to treat destitute cancer patients. We are in close contact since last 15 years. This was developed by observing a good response of Dr. Chatterjee's therapy among some of the cancer patients who were released in a hopeless condition from Chittaranjan National Cancer Institute.

Dr. Chatterjee's Psorinum Therapy was found to be very much effective for terminal cancer patients of different stages. I personally have observed many terminal patients being quite well with his therapy. In many cases it is noticed that this drug reduced growth of cancer cells in the patient's body though it is still difficult to know the exact reason for it. It can be summarised that by increasing the immunity of the body this improvement might be achieved. To be more specific, we need to have a clear idea about the molecular structure of the drug. And as such more research works are necessary in this context to know the mode of operation of the drug which has the immense potentiality to benefit more and more cancer patients. However his research works are fundamentally based on scientific approach so it is expected that the days are not far off when truth will be revealed.

In my present assignment I do spend a lot of time still now in treating cancer patients coming from distant villages in government hospital. I have thus observed and associated with the practical condition of those patients and their families. Dr. Chatterjee's overall concern about the disease and its treatment and his panoramic view about the socio-economic condition of the country have deeply impressed me all through. I have my highest

regard for him because he has always taken science as fundamental part of his therapy and his humanitarian attitude is reflected in his dealings with cancer patients. It is really amazing that in search of truth Dr. Chatterjee instead of sitting idly due to absence of huge fund required for conducting research work, dauntlessly reached to the doorsteps of the financially vulnerable section of the people with his noble heart.

In this context of my present position in my career I will be failing in my duties if I do not mention the contribution of Dr. Chatterjee to support my treatment process in both complementary and supplementary manner. I reciprocate my indebtedness to him by rendering voluntary support to the cancer research clinic at his residence. After fulfilling my official duties at CNCI whenever I find opportunity I come in contact of the destitute patients at his clinic and feel the pulse and the need of the distressful cancer victims.

As a human being I do not believe that we are to attain a harmony in our life to achieve certain goals. In case of medical practitioners and physicians if any opportunity could be provided to conglomerate the different skills available at different stream of medical treatment to find out that harmony for finding out remedy of some incurable disease, perhaps a great goal could be achieved. Dr. Chatterjee's non-conventional approach coupled with scientific attitude perhaps could open a new horizon in medical science. If Dr. Chatterjee's avowed philosophy and activities in this respect could work uninterruptedly probably we could reach the coveted goal.

Ordinary people have experienced the success of his work and will continue to do so. His work does not wait for any recognition. I will be very happy and proud to see the success of his work because I feel proud to be connected to that.

It is indeed a remarkable incident that Dr. Jeffray D. White, Director of the Office of Cancer Complementary and Alternative Medicine (OCCAM) under the National Cancer Institute (NCI) came to his house from America in the year 2004. This is undoubtedly significant that he had come far from America to much unknown ordinary lane of Patipukur, Kolkata. Evidently he would not have undertaken this major initiative had there been no positive aspects in Dr. Chatterjee's work. Today it is in fact almost a regular feature to send critical cancer patients from ASCO (American Society of Clinical Oncology) to Dr. Chatterjee's residential research clinic. This is surely an indication of his success. He has always been courageous enough to fight

with cancer and his bravery is proved by the terminal palliative centre that he has built at his residence and getting international recognition in its own way.







PROLOGUE

Dr. Subir Kumar Dutta

Professor & Head of Dept of Pathology
University College of Medicine
Dean Faculty of Medicine
University of Calcutta (Retd.)

A Holistic Approach for the Treatment of Cancer

The predictions of Leading Oncologists and Researchers all over the globe proclaim the impending growth of cancer as an epidemic within the next decade i.e. by the year 2020. Statistical data from Asian countries – China, India, Pakistan and Bangladesh are overwhelming who will be the main victims of this catastrophe. We, therefore need an uniform protocol for early diagnosis and affordable treatment of this dreadful disease. Dr. Asim Chatterjee has started working on this subject about three decades ago.



Besides narrating a few typologies of Cancer, the articles presented here are partly an autobiography of the author Dr. Asim Chatterjee and partly the history of development of his holistic approach or treatment of cancer with a simple homeopathic drug Psorinum.

In the autobiography portion he has nicely depicted the story of his empirical research on cancer. He was inducted in this area following the untimely death of his sister from cancer. He was a student of science and joined Homeopathy course to find out an alternative to traditional approach to treat this deadly disease. But he was again disillusioned when found that Homeopathic system was not based on Pathology – the branch of medicine which forms the scientific foundation of all other branches of modern science.

He started his research on cancer from 1980. During that time newer technologies like USG/CT scan have already been introduced in Western World for early detection of cancer. Histopathology and Cytopathology also improved to a great extent for confirmation of diagnosis of cancer. Physicians started using Radiation and Chemotherapy or treatment of cancer. But there were many lacunas even with those modern systems.

Dr. Chatterjee joined an NGO “The New Resource” initially and from

there he shifted to his present address 381, S. K. Dev Road, Kolkata 700 048. He started his work based mainly on pathological (tissue) diagnosis. He had a long journey to gain the confidence of physicians and surgeons of Government and Private hospitals.

He had only one Homeopathic medicine – Psorinum and mainly treated cases of cancer of lung, liver, gall bladder, stomach and pancreas mostly in the terminal phase. The research work was conducted in the state of West Bengal in India amongst 500 patients since the year 1980. The source of the drug Psorinum, a Homeopathic medicine is the alcoholic extract of the Scabies scrub/slough and is administered orally. In the initial stage the drug was administered without any supportive care like fluid infusion, blood transfusion, analgesics, bronchodilator, but the results improved markedly when both were combined.

The task of ensuring participation of both practitioners of modern medicine and this alternative system of Homeopathic medicine using a simple drug Psorinum was a herculean job and Dr. Chatterjee could do that. The activities of two diametrically opposite systems were complementary and supplementary to each other and initially cases were taken up which were beyond the purview of conventional therapy e.g. surgically inoperable/resistant to radiotherapy and chemotherapy.

Incidentally good results of this non-conventional therapy along with statistical data were published in different journals and attracted the attention of research workers in this field in India and abroad. The basic approach of modern medicine in cancer therapy is like antibiotics acting on microbial infections – a stern and drastic measure, whereas, non-conventional approaches rely on inherent healing power of human body and prefers to introduce some catalytic agents which can effectively fight out the internal disorder. Immune dysregulation and genetic mutation are likely to be the initiating mechanism.

Another important point is the consideration of very high cost of modern medicines used in the treatment of cancer. Even drugs used in the modern palliative therapy are exorbitant knowing fully well that the patient is in the terminal stage of the disease and nothing can be done to improve the condition.

Till date remarkable results have been achieved by this cost-effective non-toxic anti-cancer drug but the pharmaco-dynamics and pharmaco-kinetics of this drug is yet to be ascertained. One research group in this field in USA has taken keen interest to unveil the truth regarding molecular structure and modus operandi on this non-conventional medicine so that the present day hypothesis of this medicine may be transformed into a full proof scientific formula and accepted as a panacea or this dreadful disease.

An institute is silently and whole heartedly working in the northern part of the city of Kolkata. Patients are admitted there and treated with this so called non-conventional drug with supportive care and under the supervision of specialists of modern medicine – a unique harmonious blending. The patients with their relatives stay there with the family of Dr. Asim Chatterjee at a minimum cost. This gives immense reassurance to the patients and their family members. This is the “Critical Cancer Management Research Centre & Clinic” at 381, S. K. Dev Road, Kolkata-48. This run by Dr. Asim Chatterjee and other renowned Oncologists and Onco-surgeons practically on a philanthropic basis. Dr. Chatterjee’s son, Dr. Aradeep Chatterjee, had been to USA to get a firsthand experience on cancer management in a developed country.

NOTES FROM SOME RENOWNED PRACTITIONERS

Prof. Subir Gangopadhyay

MD Radiotherapy, R. G. Kar Medical College (Head)

I came to know that Dr. Asim Chatterjee's was on the move to cure terminal cancer patients. I was much impressed by his honest attempt singlehandedly, which is very rare among others. So I feel an urge to assist him. I tried to extend the hands of cooperation in his work. He has not come from the conventional way in the field of cancer research. But all our efforts were made to follow the proper scientific way so that the alternative ways can adjust with the conventional cancer treatment and terminal cancer patients can obtain proper benefit from his therapy.



Coming in close contact with him it was felt imperative that an oncologist is not only responsible to treat cancer patients but should also consider the socio-economic condition of the patients and his families. Patients should be provided with strong mental support too. Dr. Chatterjee was always very kind towards cancer patients and their family members. As an oncologist sometimes we feel very much helpless and are left with no other option to treat terminal patients, but to leave them for palliative treatment. We noticed that many a time Dr. Chatterjee's therapy works better for the terminal patients and it is also financially affordable. His inclination towards the serving the destitute cancer patients was quite discernible.

Here I remember one incident when we went to see a liver cancer patient living in Railway slum area at her home and she was lying on the floor and her little boy stared at his mother with pensive and vacant look. I felt sad for the child and was deeply moved by the state of his mind. I offered him my favourite pen and asked him to use this pen to become a doctor later in his life. I still remember that day. Dr. Chatterjee helped us to get the humanitarian touch of our life which is not available in any form while dealing with thousands of patients at hospital.

Many new ideas were formulated by him about the treatment of terminal cancer patients with minimum possible resources. In developed countries a lucrative business is rampant to look after terminal cancer patients and thereby

to earn a lot of money. But under his guidance we carefully shunned that way and attended the patients empathetically and with proper social commitment.

A forum was developed in the banner of 'Oncolink' at his residence (after few years it was shifted to other place) for the proper counselling of cancer patients and their family members. During our busy schedule in the outdoor hours we fail to answer the multiple queries of thousands of patients. But we felt it necessary that they are ordinary people who should be made aware of cancer in minute details for their own benefit. So an institution to arrange for social-psycho therapy for the cancer patients and their families was deeply felt. Thus Oncolink was developed with the help of some doctors and social activists, where specialist doctors could utilize the extra hours at their disposable to advise cancer patients and their family members regarding the disease. With this motto in view a unit was set up at Gandhi Seva Sangha to provide shelter to patients and their families at minimum cost. With further effort another unit was set up named 'Niramay Bhavan'.

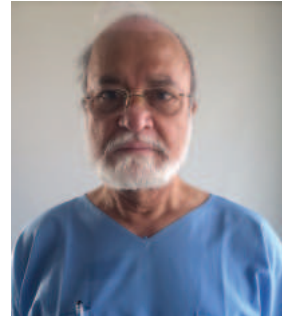
At the School of Tropical Medicine Dr. Chatterjee's therapy was mostly applied to cases of blood cancer. Occasionally he also dealt with solid cancer tumour cases. A lot of positive results came although every attempt was not successful. It was observed that many cancer patients got relief undergoing his therapy. So far my observation is concerned his therapy might have ensured some increase of immunological development in the patient's body and also his therapy might have developed to some extent an increase in the immunity power of the body parts to fight against the growth of cancer cells. But at times his therapy did not work as thought of. There may be some other typical genetic factors or some other reasons. To know the proper application of his therapy, there is a need for random clinical trial in large volume for which government initiative may be necessary.

There is no doubt about Dr. Chatterjee's devoted effort towards cancer patients. He has converted his residence into terminal centre for cancer patients where patients are not only treated but their family members are also provided with residential facilities, which is a true sign of his philanthropic attitude. Personally he is very composed, calm and never loses his patience. He trusts every person and spends a lot of money on his own for treatment of cancer patients. His wife, Ranjana Boudi, is truly a spirited and selfless lady beyond praise who is a witness of his relentless struggle against cancer and has been by his side at every step.

Dr. Dipankar Dasgupta

MD, Former Chief of Critical Care Unit,
Tata Memorial Hospital, Mumbai

It was really a memorable experience to meet Dr. Asim Chatterjee for the first time. At that point I had my assignment as the Chief in the Department of Anaesthesia, Critical Cancer Care & Pain Management at Tata Memorial Hospital, Mumbai. One day Dr. Chatterjee came with a patient's reference to the hospital at Mumbai. So far my memory goes he came along with a report of a destitute terminal cancer patient on whom he had applied his unconventional method of treatment for few months. This patient was previously released from Tata Memorial Hospital in a hopeless condition. Now undergoing the treatment of Dr. Chatterjee for six months, the pathological report showed that the patient had recovered a lot. Dr. Chatterjee wanted to know the opinion of oncologists and pathologists regarding the clinical medical report of the patient.



The striking improvement of the condition of the patient along with improved pathological findings drew the attention of all the doctors at the hospital. Enquiry was made how this spectacular improvement was made possible. Some tangent questions and caustic remarks were thrown at Dr. Chatterjee to confuse him and to belittle his efforts. Whether international protocol was properly maintained while carrying out the treatment, was asked to embarrass him. Dr. Chatterjee's answer was very polite as he said that he had only followed his unconventional method of treatment to give the patient some relief and he was glad he could save the life the patient. He was not aware if those international protocols for which he expressed sincere regret.

I was extremely thrilled to observe the entire incident and lost no opportunity to get in touch of this enthusiastic young man. I was curious to know his real intentions. Talking to him I could fully realise that his only intention was to save the patients from the disease and his humanitarian self urged on him to save the life of a person by hook or by crook, obviously on the basis of scientific reports and findings.

Dr. Chatterjee thereafter continued to go to Tata with patients from different places in order to find out conditions of the patients on whom he has applied his drug. Behind all these, he never had any economic interest. Instead, I felt he used to arrange all expenditure on his own. Many people in Tata Hospital were quite astonished to see Dr. Chatterjee's affection and passion towards cancer patients.

Whenever I came to Kolkata, I used to be very eager to know about his whereabouts, used to visit patients with him, to know about his drug. I cooperated with him when he was in Subodh Mitra Cancer Hospital and he used to consider my opinion and suggestions very seriously regarding his palliative management.

His research on non-conventional drug for cancer is thoroughly a scientific process known as Clinical Pharmacology. A specific portion of drug is given to those cancer patients who can no longer be treated conventionally or whose karnosky scale is less than 50. Of course the drug is easily applicable as it is non-toxic. Initially, however, he performed the job single handedly.

Today, Psorinum Therapy has become a major part of clinical research. He continues with his basic and clinical research with follow ups of every case accompanied by scientific investigation. It is observed that the condition of patient at every stage till the end to get a properly maintained to get well defined clinical result. He has been working on organic and metabolic function of the drug and also attempting to know the adverse effect of the drug, if any. It is needless to say that further clinical research for the molecule of the drug is necessary.

Time and again it has been found that Dr. Chatterjee's Psorinum Therapy is helping to relief pain of cancer patients and also helps the patients have food which they are unable to take. Dr. Chatterjee takes equal interest in every patient and not only treats them but also provides necessary advices to both patient and his / her family for which they depend on him and get the mental strength needed to fight cancer. He also assists them in getting all kinds of government facilities.

Dr. Chatterjee has developed a palliative management centre of cancer in his own residence, and all the members of his family in an inimitable style whole heartedly cooperate with him with enthusiasm in this brave mission. This centre functioning with the barest minimum resources is a model or the treatment of the general mass of the cancer patients of our country.

By dint of his dedication to work for humanity he has successfully overcome all the stiffest obstacles at a stretch to attend his goal. He personally comes to hospitals with patients, arranges for their cards, stays with the patients during their investigations and finally takes them back to home. His aim behind all these is only to serve cancer patients and provide them some relief.

His present objective is to know the molecular position of his drug which may require a long time. But his unflinching faith towards science never loses its momentum to find out the truth. Close friendship has developed with him since long. He treated my mother-in-law in her last days of cancer. My family members adore him very much. We wish for his success to achieve the ultimate objective.

Dr. Anup Sadhu

MD HoD Radiotherapy, Calcutta Medical College

I was transferred to Medical College in 2000 and came in direct contact with Dr. Asim Chatterjee. Many doctors of Radiological Department in Medical College used to send many cancer patients to Dr. Chatterjee. Initially, I was surprised but later on, I came to know that he treats many cancer patients through Psorinum Therapy and many people got the benefit and recovered quite well. Since then I got close to him. I observed that many doctors are sending such patients to him who even could not undergo any Diagnosis. His medicine along with all required palliative treatment is improving conditions of many patients who underwent Chemotherapy. After his treatment, reports show no further enlargement of tumour. . In many cases there has been no deterioration of patient's condition. I personally have studied many cases. It is found that Psorinum Therapy is quite helpful to keep patients stable.



When Dr. Chatterjee used to go to Subodh Mitra Cancer Hospital he was arranging for treatment and accommodation of many patients of Subodh Mitra Cancer Hospital. Economical arrangement for accommodation of cancer patients could be provided through Gandhi Seva Sangha. Even conveyance to and from Subodh Mitra Cancer Hospital was quite easily arranged. Patients who were coming from outside for Radiation could stay there. He gradually developed a Palliative Management Centre of his own in his residence itself.

Generally cancer treatment is quite expensive. This is because the drugs are quite costly. Demand cannot be fulfilled even with the government subsidy. as I came close to Dr. Chatterjee he explained me how cancer can be treated at lesser cost through Palliative management, and described how pain management and other complexities of cancer can be dealt at even 5 times lesser cost.

There are many patients who were regularly taking Dr. Chatterjee's medicines and were in good condition for a prolonged period of time. One man survived even for 4 years with Colon cancer. He used to walk from Sealdah to come here. Even patients in 4th stage of Liver, Lung, Pancreas, Gall bladder Cancer are much relieved from pain by his medicines. I have sent many of my relatives to him and they are also benefited to a great extent. One of my close relatives had Lung Cancer. It was detected at stage – IV. I asked him not to waste

time unnecessarily and advised him to follow Dr. Chatterjee's treatment. The patient acceded to my request and had neither any pain nor any agony till the time he survived.

Dr. Asim Chatterjee is a very simple person and never provides false promise to others. The most important attribute of Dr. Chatterjee is that he never accepts defeat. He never refuses anybody. He gives the correct advice to both patient and his /her family considering their financial condition. In the form of counselling he is fulfilling the task of a great social necessity.

It is unanimously accepted by all that cancer management clinic unit at his residence has become beneficial to a great section of cancer patients majority of whom belong to ordinary financial standing.

Prof. P.K. Kundu

MD Medicine

I know Dr Chatterjee since 1991-92. Detection of cancer was then quite difficult as many critical aspects could not be lightened up even by several investigations. We could not move forward much with cancer as there was many confusion. But Dr. Chatterjee's therapy along with palliative treatment was quite helpful. When a patient suffering from cancer in liver or pancreas vomit or cannot eat, he could be referred safely to Dr. Chatterjee to proceed with his therapy.

Dr. Chatterjee's palliative treatment had made huge contribution to the progress of cancer treatment. Relief from pain, ability to eat, restriction from vomiting – all are possible by his treatment. Effectiveness of his treatment is proved by its success on terminal patients.

In the initial years of 1993 we used to visit many patients with him. We, Doctors, have observed that many cancer patients were benefited by this measure due to which we always stood with Dr. Chatterjee. We believe in science, so we practically have trust on his ways. In those days, we faced many difficulties while working in School of Tropical Medicine as Dr. Chatterjee's mode of treatment did not get official approval owing to its non-conventional form. In fact, I was actually worried with all those formalities. I used to be engaged in my work and cooperated with him whenever I could. I used to encourage him a lot to continue with his work as I had the belief that his treatment would provide relief to cancer patient to a large extent. So, I requested him to make a scientific documentation of his work and assured to help him to present a paper in science congress while he continued his research work in the School of Tropical Medicine. His articles had originality and his works were fully based on science and as such we could overcome all hurdles to move forward with his work.

I gladly cooperated with him in Cancer management and supportive care and also made him aware of the different aspects of palliative treatment. He himself had enough interest to know every detail and as such today he is very experienced in this field. It is difficult to predict anything before the molecules of his drug are known but I got the experience that patients are truly benefitted by his drug.

In the year 2008, he has arranged for a centre at his residence only to deal with cancer patients. This is a real model and we appreciate his valour and courage.

Dr. Satyapriya Dey Sarkar

MS, Gastroenterologist & Gastro-Surgeon

It is a pleasure the Dr. Chatterjee and myself live in the same locality in Kolkata. Eventually I came to know that he was proceeding with his research work on cancer in the School of Tropical Medicine. Since his method of treatment was being performed in a non-conventional way, I had no idea whether there will be any occasion to work with him jointly for the treatment of cancer patients.



One day, Dr. Chatterjee requested me to visit a patient suffering from stomach problem. I agreed but was a bit apprehensive regarding the availability of necessary diagnostic reports. I was really surprised that the related papers and pathological reports were very carefully maintained and I fully realized that his method of observation was no less scientific than that of a conventional one.

This was the sometime in 1990-91 and since then I began to treat patients following his calls. I observed that he never thought about fees, his only motive was to treat the patients and offer all possible help to person. He arranged to admit patients to hospital, for blood, for government services and all other facilities to patients. Sometimes, he seemed like a social worker, rather than a doctor.

When a cancer patient first time comes to him, he explains every detail of the treatment procedure and helps the patient and his/her family in all possible way. In the present situation of Indian economy, Chatterjee's technical advices regarding utilization of government resources, the availability of private service at minimum cost and total planning to treat different types of cancer are very crucial because majority of people become bankrupt only because of misguidance. During this gruesome process he always remains very much focused to his work and continues with it with an indomitable spirit.

His passion to treat cancer patients is proved by the fact that he often visits patient's residence at his own will to find out the condition of the patient. The conveyance costs in majority of cases are borne by him.

Coming out of the mainstream and still abiding by scientific techniques is indeed a big challenge and a sign of bravery. He himself has got all the empirical knowledge and he has worked with many specialists and oncologists he accomplished remarkable ingenuity.

Once we went to visit a patient where the patient's family members were not concerned enough to meet the doctor. So it was obvious we also would not provide much time to attend the patient. I wanted to leave the place immediately but Dr Chatterjee said, "We have come to see the patient and we should not bother about the family members, neither should we get influenced by their behavior." I was a bit surprised at his words and appreciated his magnanimity with praise.

He visits to different cancer institutes for either x-ray plates or CT scan or admission of patients. The way he was and is still attached to the matter shows his grim determination. He always stays focussed about his aims and objectives and always stays cool. This is the reason behind his success. If medical course include a chapter regarding how to deal with a patient, we should first learn the lesson of proper counseling from Dr. Chatterjee. He has never asked anybody to avoid scientific methods and adopt his drug but he expects examination and review on his work also. He has developed a part of his own residence as a cancer terminal centre, this is unique. His contribution on this particular arena is tremendous. This kind of humanistic and passionate approach is very rare. His methods never exclude the main stream but always go hand in hand with it.

He mainly applies his Psorinum therapy to those patients who are either economically weak or have lost all hope from conventional methods of treatment. He has made intensive research about terminal stage of cancer patients since thirty years.

Dr. Chatterjee has amply demonstrated that in order to develop terminal centre highly efficient doctors are not always needed. It is possible with half trained and generally experienced people together under the supervision of a highly efficient cancer specialist. The terminal centre developed at his residence is indeed a model to all. His immense contribution has taught others the way to use unskilled or semi-skilled people with minimum resources to develop a centre like Critical Cancer Management Research Centre & Clinic. If such hospitals, provided with a few terminal beds can grow up in our country then that will be a great blessing for our society.

Dr. Abhijit Mandal

Asst. CMOH

Dist. Haldia & East Medinipur

It was in August 2005 when my father-in law had sudden onset of severe pain in the right Hypochondrium and was diagnosed as a case of acute cholecystitis with cholelithiasis. The gall bladder was removed by laparoscopic cholecystectomy with difficulty and specimen was sent for hispathological examination which revealed carcinoma in gall bladder and the undifferentiated cells extended up to the mucous layer. All of us with my wife became very much worried to think that he would not be going to survive beyond few months. He had been sent to Tata Memorial Cancer Institute, Mumbai and underwent a surgery there for a second time in the early September 2005. At that particular period I was studying at AIIH & PH, Kolkata. After returning from Mumbai, one day while discussing the matter amongst the friends in my Institute, one of my colleagues, Dr. Animesh Dutta suggested me to meet Dr. Asim Chatterjee, the protagonist of Psorinum Therapy which can alleviate and cure cancers to a great extent. I didn't accept my friend's statement on its face value. But when he said that his mother suffering from stage IV inoperable pancreatic carcinoma was on the Psorinum therapy still surviving and doing her usual day to day works smoothly since last eleven years I became really surprised and captivated.

I met Dr. Chatterjee with all relevant reports and documents of my father-in-law and from late September 2005 onwards he has been put on Psorinum therapy as role of chemotherapy as well as radiotherapy in carcinoma gall bladder is very much uncertain. Since September 2005 my father-inlaw has been put on Psorinum therapy and no other treatment is required for his ailment subsequently. On regular basis CA marker, CT scan and ultrasonography are being done since then which shows no abnormality. He is keeping quite fit now at his 73 years of age on this therapy and also able to perform his regular activities.

The positive outcome stated above was no doubt the result of regular administration of Psorinum therapy under the valuable guidance of Dr. Asim Chatterjee following the scientific procedure based on pathological reports.

Dr. Animesh Datta

M.B.B.S. (CAL); D.P.H. (AIH & PH)

Registrar, Burdwan Medical College & Hospital

I have spent a lot of time with Dr. Asim Chatterjee to discuss about politics, society, religion, human behavior etc. All of our discussions end with his words on cancer – the deadly disease. Presently a non-conventional orally administered drug initiated by Dr. Chatterjee has retained the smile on faces of many cancer patients throughout the world.

It was during 1996 while I was doing my house-staffship. News came from my Bardhawan residence that my mother has got Diabetes and was being treated over there. It was nothing to worry about as Diabetes is quite common ailment today. Things became difficult after 3-4 months when she got jaundice. Gradually examination revealed CA Pancreas. Many cancer specialists of Kolkata gave their words of only 4-6months remaining for my mother. Fortunately, just as a streak light from darkness, I heard about Dr. Asim Chatterjee and soon, I went to meet him. After a patient hearing he expressed his deep concern and assured to do his best. Factually my mother was suffering from stage IV Adenocarcinoma in pancreas and Dr. Chatterjee with his usual smile took up her case and continued the treatment through Psorinum therapy for a prolonged period of time. It is a great blessing and quite unbelievable that she is still surviving and doing her usual day to day work for last 14 years.

I was highly fascinated by his magnetic personality and found it quite difficult to judge his noble soul by apparently viewing his as a doctor. Curiously enough when cancer treatment meant, it is beyond the reach of the common man, Dr. Chatterjee by dint of the uniqueness of his research programme reaches to the slum of the pauper in quest of knowledge.

His research on cancer has taken him to the heart of many people as a savior. He is the true worshipper of science. We know how much difficult it is to continue with research work in a Third World country like India. But we believe that by virtue of his humanistic approach and indomitable spirit he will be able to overcome all odds and to reach his targets.

Dr. Asim Chatterjee, according to me, is quite ahead of time, so his appropriate evaluation will perhaps be done correctly by the next generation only.

Dr. Hiranmoy Mukherjee

MD CAL, Ph. D.

Ex HoD Dept. of Entomology

The School of Tropical Medicine

I have seldom found such a delicate research worker like Dr. Chatterjee in my life. His enthusiasm, initiate, perseverance and tenacity to act against all odds simply overwhelmed me. Incidentally I came to know that when Dr. Chatterjee for the first time approached Dr. A. K. Hati, the then Director of the School of Tropical Medicine to cooperate in research work on cancer, he was bluntly refused. The proposed drug Psorinum itself appeared to Dr. Hati of no use and completely bereft of science. Later on, it was noticed that Dr. Chatterjee was making direct contact with the terminal destitute cancer patients and desperately trying to continue their treatment with drug Psorinum. In this process he had attained partial success in certain areas like relief of pain, reduction in vomiting and restoration of their ability in taking food. This encouraging and positive response among the patients drew special attention of many of us who were attached to the School of Tropical Medicine and requested Dr. Hati to change his mind and to cooperate with Dr. Asim Chatterjee to work on terminal cancer patients.



Since then we became intimate friends and had regular contact with each other. Later on he got introduced to Manju Dutta Chowdhury (Dept. of Haematology, School of Tropical Medicine) and she was willing to work with Dr. Chatterjee and as such he came across many doctors of the School of Tropical Medicine and could deal with many cancer patients. But unfortunately a section of doctors had opposed the unofficial involvement of Dr. Chatterjee in process of clinical trial on the destitute cancer patients. A daily news paper published a report about these unofficial activities of the School of Tropical Medicine which led to bitter atmosphere in the institution and situation forced him to stop his research work in the end.

Although during this time several papers were published jointly regarding Dr. Chatterjee's Psorinum therapy in the Bulletin of the School of Tropical Medicine and also in the Indian Science Congress with the assistance of Dr. Hati and Dr. Subir Dutta. Then we went to the Medical College, University College

of Medicine, Department of Chemical Biology to continue his research work. Thereafter he went to Tata Cancer Hospital, Mumbai, to fulfill his research objectives.

While working in the School of Tropical Medicine we went to the residences of different cancer patients for palliative treatment. Dr. Chatterjee had a benevolent set of mind regarding all these. Unlike other non-conventionalists he was very much particular in evaluation of condition of the patient by maintaining pathological reports meticulously which obviously indicates his scientific bend of mind. Dr. Chatterjee's therapy expanded the life-span of many bed ridden cancer patients and also relieved pain to a large extent. Patients who cannot eat, who vomits regularly, is being cured to that extent that the patient was able to take food and vomiting tendency also reduced.

At present, he has made a palliative management and terminal centre for cancer patients at his residence. After my retirement I have been engaged with research projects of Critical Cancer Management & Research Clinic. Now he directly applies his therapy on cancer patients. And in many cases get good result. Still he has a long way to go. Especially it is necessary to identify a molecular structure of the drug Psorinum which is instrumental for the shrinkage of malignant tumours. Under his leadership with the judicious combination of palliative treatment with the drug, perhaps a glorious chapter will be opened in near future on the treatment of cancer.

Dr. Sudin Bhattacharya

Senior Scientific Officer (Senior Assistant Director Grade)

Dept. of Cancer Chemoprevention,

Chittaranjan National Cancer Institute, Kolkata

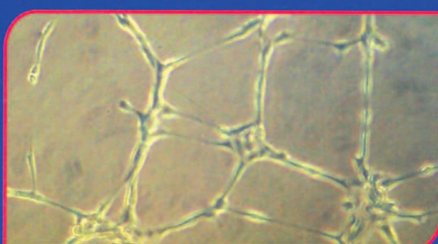
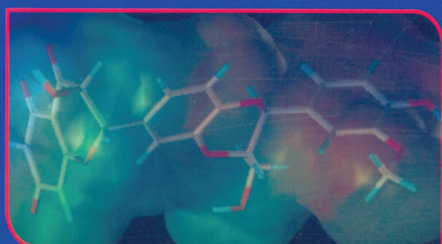
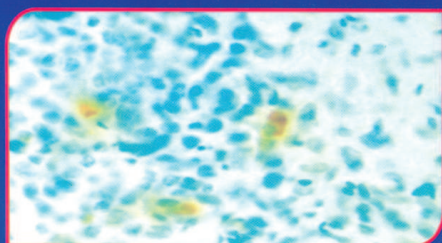
Psorinum Therapy is a well documented treatment and has now been clinically tested and proven in Kolkata and India. It has been observed that the non-toxic Psorinum has shown encouraging result in treating several types of cancer. The most important thing that needs to be mentioned here is that Psorinum, in over the years, has proved and secured its place as the complementary and alternative medicine in the treatment of cancer patients. One of the reasons behind that could possibly be that the drug was chosen to treat cancer that is pathology-proven.



But we are not physicians here. We the scientists are more concerned about the scientific development of the drug.

Usually the scientific work is done first to prepare the ground for clinical trials. But in this case the clinical trials prepared the ground for future scientific research and development. Another factor favouring the use of Psorinum therapy is its non-toxic character. This drug has been prepared from the extracts of a specific kind of mite. I admit that scientific works are generally very complicated and time-consuming. However, in the very nascent stage the work has gained significant prominence. In the year 2008 we had sent a paper titled “Non-conventional cancer treatment Psorinum therapy becoming persuasive to the scientific community” which was presented by Dr. Aradeep Chatterjee at the Jawaharlal Nehru University, New Delhi, in my presence. Later on this paper got published in the ‘International Symposium on Novel Strategies for Targeted Prevention and Treatment of Cancer, December 19 – 20, 2008’, by Jawaharlal Nehru University, and it was awarded the best work that year and gained much appreciation.

**International Symposium
on
NOVEL STRATEGIES FOR TARGETED PREVENTION
AND TREATMENT OF CANCER
December 19-20, 2008**



**School of Life Sciences
Jawaharlal Nehru University
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line Panc1-G from the human pancreatic cancer cell line Panc1 by prolonged exposure of parental cells to increasing concentrations of gemcitabine. *In vitro*, the IC₅₀ increased from 0.050 μ M in parental Panc1 cells to 10 μ M in the resistant variant. In order to examine different proteins involved in creating resistance to Gemcitabine, we have analyzed Panc-1 and Panc-1G cells by Real-time PCR analysis and High Content quantitative Image analysis of proteins on CellomicsVTi array scan platform. The results revealed that over expression of ETS-1, RRM-2, Cox-2, MMP1, Bcl-2 and cyclinD1 in Panc1-G cells when compared with parental Panc-1 cells. The expression of ETS-1 was silenced in Panc1-G by ETS-1 specific SiRNA. The results reveal that ETS-1 silencing in resistant Panc-1G cells showed significant down regulation of ETS-1 at 48 hrs after transfection while control scrambled SiRNA did not show any significant change. After 48hrs ETS-1 silencing we have also noted significant down regulation of Bcl-2, MMP-1, CyclinD1, Cox-1 and up regulation of Pro-apoptotic protein BAX and CytochromeC levels in ETS-1 silenced Panc-1G cells. These findings may suggest that ETS-1 is a key regulator of cell cycle progression, invasion and resistance in pancreatic cancer.

Keywords: ETS-1, gemcitabine, RT-PCR

P-34 Non-conventional cancer treatment psorinum therapy becoming persuasive to the scientific community

Aradeep Chatterjee, Sudin Bhattacharya, Ashim K. Chatterjee, Jayanta K. Das, Pramita Chakraborty, Prasenjit Ghosh, Joydip Biswas

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Present observational clinical trial was conducted to study the effect of an alternative cancer treatment called Psorinum Therapy (very popular in Kolkata, India) for the treatment of oral, lung, liver, gall bladder, pancreatic and stomach cancers. A pre-clinical trial on mouse model bearing EAC cells has been conducted to know the mechanism of the drugs of Psorinum Therapy. The drug Psorinum (an alcoholic extract of scabies, scrub, slough, pus cells) was orally administered (0.01ml to 0.02 ml /Kg Body Weight/ Day in an empty stomach) to 245 (23 oral, 62 lung, 32 liver, 40 gall bladder, 44 pancreas and 42 stomach) cancer patients along with allopathic and homeopathic supportive treatments. Conventional cancer treatments could be initiated due to financial constraint, late advance stages of the disease and due to poor general health performance status of the patients (Karnofsky: 50 or below and ECOG: 3- 4). Among 245, 11 (4.49%) diagnosed at primary stage, 73 (29.8%) diagnosed at intermediate stage and 161 (65.71%) diagnosed at advanced stage. Among the 245, complete tumor response occurred in 54 (22.04%) cases and partial tumor response occurred in 78 (77.96%) cases. 200 (81.63%) have overall 1-Year survival, 156 (63.67%) have overall 2-Year survival, 140 (57.14%) have overall 3-Year survival, 121 (49.39%) have overall 4-Year Survival and 101(41.22%) have overall 5-Year survival. In addition qualities of patients' life were improved in majority of the cases. Significant results of Psorinum therapy have also been observed *in vivo*. The findings indicate that Psorinum Therapy is very promising for the treatment of oral, lung, liver, gall bladder, pancreatic and stomach cancers. Patients report no side effects from this therapy. The Psorinum Therapy is quite cost effective providing additional practical feasibility in the prevalent socio-economic scenario of the developing countries like India. Whether this treatment protocol can be accepted for further scientific research and can be replicated to cure or control these types of cancer now need to be analyzed.

Keywords: Psorinum, homeopathy, CAM, EAC cells

Dr. Amitava Chakraborty

MD, Radiotherapy, Associate Prof.

R. G. Kar Medical College & Hospital, Kolkata

I know Dr. Chatterjee since 15 years in the field of cancer. As an oncologist I have observed many of his cases. He treats patients in whom CT and RT are not applicable; his methods have relieved pain of many severely ill cancer patients. Efficacy of Dr. Chatterjee's Psorinum therapy has indeed been proven.

A large number of cancer patients of our country are not able to afford conventional expensive cancer treatment. In this context, there is no doubt the efficacy of Dr. Chatterjee's Therapy. Earlier he visited different patients at their homes or in the hospitals. But now he has brought his scatteredly treated cancer patients to his centre of Critical Cancer Management & Research Clinic where everyone is treated equally. Atmosphere of his centre is very homely. It is necessary to know the scientific molecular structure of his drug and for that reason collaboration with a large unit is urgent in order to carry forward his research work.

Methodical clinical trial is not a work of a single day. It is a long process. At present at his clinic random trial of terminal patients is no more difficult because it is proved that the drug has no toxic effect. As such collaboration with a large unit will help the centre of S. K. Dev Road, Kolkata to continue its research work successfully. Critical Cancer Management & Research Clinic continues to play a vital role in cancer palliative management. In future if any University Research Institution comes in forward then it would be easier to carry another part of research works.

It is quite astonishing to think that he continues with his terminal centre with such a minimum resource. Undoubtedly Dr. Chatterjee's success can be attributed to his courageous moves, innovative initiatives and pragmatic approach based on reality.

PRELUDE

Golok Chandra Roy

I, Golok Chandra Roy, had got involved in the 1971's Bangladesh's struggle for freedom at a very young age. There, fortunately, I got introduced to the then army-chief General M.A.G. Osmani who took me out of the front and appointed me his P.S. I stayed with him till he breathed his last. After that I joined a government bank as an officer. But soon enough I came in contact with Prof. Mohammad Yunus and he offered me to join him as his P.S. I gladly accepted his offer. I had been with him for 30 years. I have seen how such a small initiative took the shape of a large micro-finance organisation. I was associated with him even after he had won the Nobel Prize in the year 2006. In the early life when Prof. Yunus used to say something to the new joiners of the organisation, they hardly used to understand this perspective. But now things have changed. Now people from all over the world eagerly wait to attend his seminars and listen to his lectures.

In the year 2007 I accompanied Prof. Yunus to his visit to Kolkata. This was the time I met Dr. Sisir Dutta and Dr. Asim Chatterjee. Dr. Sisir Dutta did not know Dr. Chatterjee up until that time. Dr. Sisir Dutta's domestic help was suffering from gall bladder cancer then. Many renowned institutions and oncologists had raised their hands off the case. At that time Dr. Jaydeep Biswas introduced Dr. Sisir Dutta to Dr. Chatterjee for any advice and assistance. Dr. Chatterjee gladly accepted the case and now it has been nearly ten years that the lady is alive and is leading a cancer-free healthy life. This entire episode brought me and Dr. Chatterjee close to each other.

Our association did not continue for long as I had to move to China following my professional commitments. The Chinese Government was very much attracted by success of the Grameen-bank model pioneered by Prof. Yunus and had asked him to send an expert to their country to establish a similar model there. Prof. Yunus at their request asked me to go to China with this responsibility. I was in China for many years thereafter.

I am not a physician. But apart from treatment I have observed the social aspect of cancer which has attracted me a lot. Dr. Chatterjee apart from treatment also takes initiative towards the employment and rehabilitation of the families of poor cancer patients. The kind of scope his module provides to overcome such critical issues has startled me and has forced me to think about all that is going on around me twice.

In order to initiate any new work, first we have to think about it in

details, then work towards its execution. During this stage we have to take many steps. Some of them might go wrong but our ultimate goal should be fixed. On certain occasions it might take some time to reach our predetermined objective but with right intentions and right people accompanying one will sooner or later achieve success. I have seen General Osmani in Bangladesh's struggle for independence. I have seen Prof. Yunus bringing in the socio-economic transformation in Bangladesh. Today I see Dr. Chatterjee doing a commendable job within his small initiation, Critical Cancer Management Research Centre and Clinic (CCMRCC). Seeing him doing all this single-handedly I wish to work with him someday and contribute towards the well-being of those unfortunate families who have lost their dear ones and are trying to overcome their financial limitations. With this thought I wish Dr. Chatterjee and his family, specially his son, Dr. Aradeep Chatterjee, all the luck, prosperity, good-health and success in life.

EXORDIUM

Piyanka Mohanty

M. Sc. Economics C. U.

Little I had heard of Dr. Asim Chatterjee when I first met him on 5th June 2015. I visited Dr. Chatterjee in regards of my ailing uncle who was suffering from pancreatic cancer. He had come down to Kolkata from Rourkela, Odisha, after referred by a doctor from AIIMS, Bhubaneswar. He was admitted on the same day and was under Dr. Chatterjee's treatment at Critical Cancer Management and Research Centre for six days. But then he had to leave due to some professional commitments.

During my uncle's stay here I read about Dr. Chatterjee and Psorinum therapy through various sources. I learnt that he has been successful in regressing cancer in many terminal stage cancer patients. Every day I used to visit my uncle at CCMRCC and used to spend hours talking with Dr. Chatterjee regarding my uncle's health and on the subject of cancer. In due course Dr. Chatterjee told me about the severity of the disease and its impact on our economy. Being a student of Economics I started taking deep interest on the subject. The day my uncle got the discharge from CCMRCC Dr. Chatterjee called me and told me that he was facing some difficulty in writing a portion of this book which focuses on the socio-economic aspect of our society and within the next ten years how the spread of cancer is going to affect us and our economy. He asked me to put on paper our country's current health scenario with the backdrop of cancer. My first question was, whether he thinks that I would be able to do justice with this job. He said if not me nobody else can since I have seen someone very close to me suffering from the disease and assured me all kinds of guidance and support.

There has been times when I have made mistakes but Dr. Chatterjee always took it with a smiling face and made me understand the complexities of the subject. In the mean time I also got a job opportunity, however, I assured Dr. Chatterjee that till the time his book gets published it would be my first priority. It has been more than a year now that I am working with Dr. Chatterjee not only on the socio-economic aspect of cancer but also on the technical part and the presentation of the materials. During the initial days I used to sit with him taking down notes and I have also come across many oncologists and got the privilege to ask them questions and take their interview like Prof. A. K. Hati, the former Head of the Entomology

Dept. and ex-Director of the School of Tropical Medicine, Prof. Anup Majumdar, Former Professor of Radiotherapy, Radiotherapy Dept. B. S. Medical College, Dr. Hiranmoy Mukherjee, former Head of the Entomology Dept. of the School of Tropical Medicine, and Dr. Asoke Bose, Secretary of Subodh Mitra Cancer Hospital.

I sincerely believe that Dr. Chatterjee's efforts will go a long way in the field of cancer research and treatment in the near future.

PROLUSION

BETWEEN SLEEP AND RISE

Dr. Asim Chatterjee

This book contains my 35 years long journey, a no-one from nowhere to someone from a distinct sphere. Several years ago I had alone embarked on this journey facing many obstructions, both human as well as technical. Most of my youthful days got wasted since I was not being able to decide what to do and what not to do. There was a time when I also thought of writing poems. But after sitting for several days no words came in my mind and I realised that I was not meant for it. Since 1978



I started taking things more seriously. One of my friends was working on Sectarianism and soon I also became excited and started researching on this topic. I also enrolled myself in the M.A. course. This was the time when my elder sister was diagnosed with cancer. We all tried so much to save her but as destiny had decided upon something else; she lost the battle with cancer. Soon I found myself surrounded by so many helpless people struggling for their life. The scenario was very disheartening for me and my elder sister's death used to haunt me. I was then determined to do something for them; hence, irrespective of the fact that I do not come from a medical background I started studying medical science. In due course I found brilliant guides in Prof. R. S. Bhakta, Prof. R. N. Brahmachari, Prof. Anup Majumdar, Prof. Subir Dutta, Prof. Subir Ganguly, Dr. A. K. Varma, to name a few, who practically walked with me through rains and storms. I also found a lifetime friend in Dr. Hiranmoy Mukherjee.

Despite the variety of problems, and hence the variety of strategies for tackling these problems, certain common themes and underlying principles emerge. It is these principles, requiring an understanding of both socio-economic and biological factors, which it is hoped will emerge in this book. The added responsibility of writing a book summarizing the lessons that I have learnt was also very stimulating. I decided to talk about my 35 years of research on cancer and my family, friends and colleagues who supported me during those tough times.

Around 8 years ago, a prominent personality from the medical

fraternity of USA visited our residence-cum-facility-centre after learning about us from different journals and articles. He had told me: *There is a truth underlying in this work. Usually a scientist's calibre is measured by the number of publications he has by his name. But in your case a multi-directional work has sprang up without much publicity. The concept with which I had come here, in one day my perception about you has completely changed. Your research is far ahead of this time.*

I had tried to preserve all the case reports of the patients I had treated. Unfortunately, due to poor management during those times many valuable documents got destroyed. Also I would like to confess here that I have to run a hospital and also look into its administrative aspect. As a result I did not get enough time to dedicate towards the writing of this book. It took me two years to complete this book. But I feel it was all pre-destined.

I have always tried to select a problem whose solution would be significant. On the other hand, the problem must be one that could be solved with the means at one's disposal.

The earlier version of this book, 'A Total Strategy Against Cancer – Studies in Diagnosis, Treatment, Rehabilitation and Terminality', was my first attempt as a writer which came out in 2013. I would like to mention names of four people whose encouragement gave shape to this book – Prof. Subir Kumar Dutta, Pushpendra Krishna Bhowmik and Dr. Jaydeep Biswas.

I have tried to make this current book 'The Management of Cancer in Totality – India Can Take a Lead in Theory and Application' material-wise more rich and presented it in laymen's terms as much as possible. I have also included a few topics of the previous book here as well. Innumerable support poured in from my son, Dr. Aradeep Chatterjee who is also the co-author of this book and was deeply involved in the planning, compilation and presentation of the book, and my daughter-inlaw Mrs. Swarnali Chatterjee. My special thanks to my wife, Mrs. Ranjana Chatterjee, for supporting me throughout the time this book was under-making, both mentally as well as financially. My heartfelt gratitude and sincere thankfulness to Mr. Shankar Chakraborty without whose selfless efforts this institution would not have gained this ground of reality. He is no less than a family to me and I would always remain grateful to him.

The people who helped and encouraged me to write this book include Prof. Anup Majumdar, Prof. Amiyo Hati, Dr. Hiranmoy Mukherjee, Subhodeep Hazra, Piyanka Mohanty and most importantly Mr. Prithwipati Chakraborty. Mr. Chakraborty and Piyanka both have worked on the socio-economic aspect of this book and also guided me on many occasions. I am

short of words to thank Mr. Chakraborty for taking out so much of time from his busy schedule and also introducing me to Mr. Sanjib Dhar, who helped me in publishing this book many ways. I would also like to thank my hospital staff for being so patient with me and being by my side through many ups and downs.

I would like to express my heartfelt regards to Dr. A. K. Varma. He encouraged to pen down my thoughts, treatment methods and a few case studies and assured me to document the same in the next Congress session. He had spent days after days with a view to documenting my research studies. He guided me to create such a platform as would be accessible to many. I would like to acknowledge the co-authors of the two papers for their patience, time and unimaginable support in my favour, and the utmost honesty they have shown towards the project. It had taken me six months to write the paper but it took around a year to decide upon a title. Dr. Anup Sadhu and myself titled the paper as “The Management of Cancer in Totality – In Theory And Application”. But Dr. Varma modified it to “The Management of Cancer in Totality – India Can Take A Lead In Theory And Application”. He once wrote to me: ***I bless from my heart. I am confident that this work would one day lead the entire world to create a cancer-free tomorrow.*** This paper was later published in the book “Tobacco Counters Health Vol – 4”. A few people raised questions as to appropriateness of the title but I took full responsibility and got the paper published. Many renowned personalities were engaged in this work and the output we received was highly valued. As already mentioned, two of my papers require special mention. They are “Non-Conventional Treatment of Tobacco Related Cancer Gradually Gets Right Perspective Through Psorinum Therapy” and “The Management of Cancer in Totality – India Can Take A Lead In Theory And Application.” Dr. Varma told me that the next generation working on Cancer treatment and cure would draw inspiration from these two papers.

I would like to thank Mr. Pushpendra Bhowmik. He would always hold a special place in my heart. When I first started putting down my thoughts on paper it was Mr. Pushpendra Bhowmik who led me through all the details and intricacies of writing a book. The people who have acted as my pillar of support through the rough times include Dr. Subir Dutta, Prof. Amiyo Hati, Dr. Hiranmoy Mukherjee, Dr. Subir Ganguly, and a special mention of Dr. Jaydeep Biswas for his personal and institutional support. The people who helped me in my research are Prof. R. S. Bhakta, Prof. R. N. Brahmachari, Prof. Anup Majumdar and Prof. Dipankar Dasgupta. Mr. P. Chakraborty has cooperated with me every time whenever I needed him. My heartfelt gratitude is on record towards Gautam Saha and my

scientist friends, Dr. Gautam Mukhopadhyay and Dr. Ashis Mukherjee who had helped me at a certain point of time. I feel privileged to receive such unending co-operation from these personalities.

Dr. Gautam Mukhopadhyay was the person because of whom I became a part of the Tata Memorial Hospital. He overlooked the existing practise, made me a part of several surgeries and enlightened me on various practical aspects of surgery. This experience I would never forget in my life.

From the very beginning I had a vision to do something big and beneficial for my own country. I had started the preparation a long time back. A few mistakes I had made in my life, but very soon I realised it and stepped back. However, this helped me a lot in my subsequent research and decision-making process.

I wish to make this book in six volumes. Meanwhile I have arranged all the materials, some old and new case studies, many research papers for reference and recollected some of my experiences and association with some renowned personalities belonging to this field as well as some national and international accolades. I had introduced the publication of my works in my previous book titled “A Total Strategy Against Cancer – India can Take a Lead in Theory and Application”. Here I present some surprising case study reports, some cellular level work, some reality oriented research, and would uphold the socio-economic scenario of India in much detail. We would also look forward to find a resolution to the existing issues.

I propose to pen down 5 more volumes of this book with special emphasis on each type of cancer. I have decided to write on Lung Cancer in the next volume which will be more technical yet simplified for the general masses to read and understand. Also much more focus will be given to my son, Dr. Aradeep Chatterjee's work on cancer and our recognition by international forums like ASCO. I would apologise if someone comes across any mistake since it is totally unintentional.

My Confessions

It is in fact a riddle how I myself having so much scarcity of resources dared to enter in this venturesome project and preferred to continue my battle against Cancer in on unknown path singlehandedly.

Many of my friends find in me a person of bohemian type but on deeper introspection I appear to have possess a striking balance of both positive and negative aspects of mind, while I started my eventful journey with Psorinum Therapy. Gradually I could analyze and identify both of my strengths and weaknesses which I would like to state frankly.

- Inadequacy in higher scientific knowledge and inexperience in handling cancer patients were my first drawback.
- Absence of proper scientific equipments and research gadgets was another major shortcoming.
- Minimum laboratory-support and medical dispensary was not available.
- Attachment to or smooth and welcome entry to any Government Hospital or Cancer Institute was not for me.

In spite of all these serious drawbacks or obstacles my conviction and commitment to carry forward this stupendous task was made possible perhaps due to presence of some positive factors which was instrumental in building self-confidence within myself.

- The first one is my sound health, enthusiasm and utmost devotion towards achieving the goal and unstinted faith in scientific study.
- The second one is the economic support that I could derive from my ancestral property. As a security measure against financial odds, my father who was well aware of my indifference towards monetary expenses and incapability of managing financial affairs wisely, provided a trust with sufficient fund which could be utilized to carry forward such expensive projects.
- Owing to strong desire to work for the cause of the common people I secretly got myself associated with a political movement during the period of 1970 – 72. But during these two years the bitter experience for many untold incidents emerging out of hard reality intertwined with this movement led me to ultimately dissociate myself from that path and prompted me to find out a way to serve the real cause of the common people. In the latter part of my life however, these diverse experiences helped me immensely to face many critical problems and to overcome difficult situation skilfully.
- The fourth positive factor for me was the unsatisfactory progress in the world-wide treatment of cancer in the conventional way. In spite of introduction of big-budget research programmes in foreign countries coupled with application of advance scientific technologies, treatment of cancer, throughout the world, could not make much headway in curing this disease. It is apparent that till date a large area in the domain of cancer is lying in darkness as regards the virtual nature and treatment of this disease. This phenomenon provoked me to think that in a completely dark road having no speck of light the

persons even with full eye-sight is never better placed than a blind man. Rather a blind man in this situation moves faster, smoothly and comfortably as he is accustomed to such situation.

This deep understanding encouraged me to tread on a way which was so difficult to move without having modern and advanced scientific apparatus. However, while conducting research work with Psorinum Therapy the main principle of result-oriented practical scientific approach through three distinctive steps of experiment, observation and inference was never lost sight of.

I feel it imperative to deal with the problem of realistically tackling the management of cancer in the Indian sub-continent particularly with reference to its socio-economic scenario. Among the cancer patients who are sufficiently rich they can take the opportunity to be treated in conventional method even in foreign countries. The next group who are financially sound belongs to higher echelon in the society, can afford to pay exorbitant cost in the big hospitals in Delhi, Mumbai, Bengaluru, Hyderabad etc. Residents of Kolkata depending on their financial capability would at first try to get admission in sophisticated hospitals, but when the treatment cost would go beyond their reach, most of them would be left in the lurch. So will it not be prudent to chalk out a programme of treatment of cancer realistically on the basis of financial capability and socio-economic perspective of our country? I admit, these issues always haunted again and again and acted as a motivational factor to carry out this tortuous task with all calm and confidence.

In the present context, sometimes it may appear quite unbelievable to imagine the existence of such a Cancer Centre of ours with ten beds and with operational facilities along with its research wing, which has been functioning smoothly for years together by observing extreme frugality and with strikingly meagre resources. Practically the clinic is run with the barest minimum requirements regarding 4 Ms i.e. “Man, Machine, Material and Money.”

The integrated efforts of all persons engaged in the clinic in rendering service to the patients have perhaps made this impossible event possible. All the patients whether they are at terminal stage or not are getting necessary care. It has always been taken up with a missionary zeal to pay proper attention to the patients as well as to their family-members and to maintain at the same time how to show due respect to them especially in

the last days of life. The collective wisdom to do away with all obstacles transcended the entire thing to a great height.

At the present stage of my journey I have no hesitation in conveying my feeling that this journey which began to see an end of Cancer has become so much exhilarating that I have no words to express. After so much ups and downs, twists and turns, success and failure I perceived that the only thing which I have to perform continuously is to keep the flames on. I am a firm believer in God and all the time in my journey I sit on my knees and pray: "Lead the kindly light."

Brief Synopsis Of The Book

I have come across many facets of human life, issues and challenges. The results I have obtained, I am sure, will definitely help many people to explore a whole new path and open unknown avenues for the treatment of Cancer in future. The satisfaction I get from this is something more meaningful and precious than any academic degree, award or recognition. I am looking forward to many more challenges that may usher in my way in the coming days.

One of my friends, Prof. Jyotirmoy Chakraborty, former Professor in the Physiology Department of the Presidency College (now Presidency University), once said to me: ***I have worked on Cancer for so many years in laboratories. I along with my colleagues have worked so much on Cancer that we are capable to outdo any oncologist on any day. However, when we find ourselves on the practical side of it, we put our heads down and fold our hands while visiting the oncologists, and accordingly accept and do whatever they say, and act.*** He also said to me: ***You have walked through all kinds of roads, and ditches, and if you try, you can write down all your experiences taking into consideration all practical aspects of Cancer and lives of people affected by the disease, in an authoritative way.***

It was Prof. Jyotirmoy Chakraborty who first inspired me to pen down all my experiences and whatever knowledge I have acquired, and present it to the public. In this book I have tried to present an outline of my long tireless journey towards life out of Cancer and I wish to discuss more on this subject in the chapters to follow.

A few days ago when Prof. Jyotirmoy Chakraborty visited my medical facility Centre, I discussed with him regarding the book and also what plans I had in store for the future. He became quite surprised and frankly admitted that he had not at all been serious when he had asked me to write. Now he became very happy to know that I would be sharing my experiences with the masses and said that it was required.

In this book besides outlining the detours of my journey to search on effective low-cost therapy for cancer, I have tried to read between the lines the socio-economic picture of our society. I entered into the medical profession as a non-professional medical practitioner. I had to work with people who belonged to the lowest strata of the society. I never considered myself to be able to do something spectacular for the common people nor did I mean to start a mass awareness campaign, but the work I have done till now has been entirely for the poor, staying in the midst of the poor and terminally or critically ill patients. Today, whatever I say may seem hilarious to many. However, after the publication of this book I am sure people would gradually accept my credibility and as time would pass, they would also acknowledge the intensity of the situation. As I have already mentioned, experts are predicting that by 2025 i.e. within the next ten years, cancer would affect almost each and every household. Many many households together build a nation, which means, by 2025 our entire nation would be affected either directly or indirectly. Under this scenario we no longer would require a third world war for mass destruction. The number of lives a third world war is expected to take away, cancer alone may surpass that number. In a nation like India where population is increasing with each passing second, a fall in population may not be an engaging concern from the society's point of view, but losing lives due to cancer would eventually swipe away valuable human capital resources that are available to any household. This is definitely a matter of serious concern. In this backdrop what kind of role we are expected to play needs to be visualised and discussed.

We also have to think about those people who are at the potential risk of becoming a victim of cancer. For obvious reasons, middle-class people cannot afford to be emotional at this point of time. We have to look into both the scenarios for the overall well-being of the nation.

The pace at which cancer treatment is progressing, it is very difficult to predict now that in next fifteen years how much successful and affordable it would be for the common people. So, as I have already said, in this nation where nearly 85% people would lie outside the scope of cancer treatment is a haunting phenomenon that is gradually coming true. It is time to rethink about those 85% people all over again in a more realistic way. Here I would like to quote Prof. **Amar Bhaduri: A nation's problems would only be resolved in that country itself with its own materials and human resources, otherwise not.**

We have to declare a war against cancer. This was proposed by Prof. R. N. Brahmachari. I diligently started working towards it and eventually was able to construct a successful model. This model is our residence. My family-members have supported me and also contributed a lot towards

the overall development of this facility as well as the well-being of the patients treated here. My intention from the very beginning was to go beyond the horizon and deliver. In my journey I may have bypassed the conventional laws and I will continue doing so in order to obtain life-saving results for the humanity. For achieving this goal we have come out of the bookish methods. In my very modest opinion, the planning should be such that it would be future-oriented and more close to reality. We have to treat the patients on a case-by-case basis keeping in mind the socio-economic status of the family as well as their educational background, life-style and proximity of the patients' residence to the city. In this backdrop, we have to identify the disease, identify its type depending upon the occurrence in the body, and also work towards the final result together.

The Cancer treatment model I have explained in this book, I believe, would serve millions in the long run. The results that I have observed till date and would be done in future will be provided in details in the forthcoming volume.

Background of writing this book:

In order to give a shape to my ideas and documentation of certain facts I felt the need to write a book to share my findings and itinerary of my journey so far. Many people told me that the development of my cancer treatment therapy was very haphazardly documented so far. There was a need to arrange and bring it together for someone to understand.

Prof. Dipankar Dasgupta, Prof. Anup Majumdar and Prof. Jaydeep Biswas said that it was very important to write a review of the research works before writing a thesis book. Nobody before me had either done any informative and experimental work about Psorinum Therapy in the context of cancer treatment or worked on the source of the drug involving the pharmacokinetics and pharmacodynamics. This had given me a good opportunity to showcase my works on cancer since the 1980s. Accordingly I prepared a 10-page synopsis on the subject and showed it to Prof. Dipankar Dasgupta, Prof. Anup Majumdar and Prof. Jaydeep Biswas. They were very impressed at my effort and encouraged me to go ahead with the idea of the book though they admitted that they would assist me in whatever way possible. It was possible to compile and write down the book after one year of non-stop effort. I am grateful to Dr. Subhodeep Hazra and Dr. Sudin Bhattacharya, of CNCI, for their contribution. In this book we have tried to bring in various facets of socio-economic aspects and human nature. Piyanka helped me a lot in bringing these pieces together. Piyanka and I have tried to write it in simple language for the readers to understand the situation and accordingly to go about. If anyone outside the scientific community wants to know on the subject of oncology, this book may act

as a help. In this book, I have depicted almost all the experiences I had in my journey like a toddler's hop to the sky of infinity. In every hop, there was lure of life and in the next, the ask me or awe of death or losing everything. A few of my articles, nationally as well as internationally acclaimed, have been re-produced in this book. Days of my marathon journey towards the goal, despite not being an athlete, and stop-overs here and there under the sacred roofs of learning like Indian Association for the Cultivation of Science, the School of Tropical Medicines and others have been described here. People ask me about myself. This is myself as depicted here - nothing more, nothing less. I prefer to remain as non-descript as far as possible.

My Views

Amidst many criticism, odds and demoralising humiliations, I stood firm, composed and determined, and ultimately become able to serve around 2500 patients in my medical facility Centre. I was able to do this only with the firm resolve and mental strength to overcome any hurdle whatever would come in my way. At CCMRCC, with the West Bengal Government's recognition, renowned doctors make regular visits, patients undergoing indoor treatment are kept under 24×7 hour's observation. With barely minimum resources this treatment facility has shown tremendous potential and results not observed earlier. This facility acts as a ray of hope to thousands of poor, helpless and hapless victims of Cancer. Many foreign oncologists and scientists want to replicate this treatment model in their country. I appreciate people's acceptance towards us and also acknowledge the confidence they have reposed on us and this facility. I have decided to present this facility's treatment-protocol in the volume to follow.

One day something that started as a research-oriented work, today it has attained the shape of a new treatment module altogether. We have seen and treated thousands of patients in these 35 years. Our hospital records speak in volumes about the thousands of patients treated here and benefitted, both national and international. We have preserved the HP reports, FNAC reports, CT Scan reports etc. and the progress and future development reports of the patients in consultation with experts for years now. The records also point out how we handled the problem with the minimum disposable resources that we had in hand. I would also like to share my deep regret that some of the valuable case studies were destroyed in the process and many important letters got defaced. Still I have tried to provide as many case-studies as possible in this book so that any scientist or specialist physician going through them would understand under what difficult situations I had to work in order to reach the pre-defined goals and would acknowledge the contribution of Psorinum Therapy in the treatment of cancer. It would not be possible for anyone to ignore the facts as many renowned personalities and institutions are associated with these case studies and the research behind. Most of the cases treated here

have been institutional cases i.e. patients coming from institutions like SSKM, Chittaranjan National Cancer Institute, Calcutta Medical College, R.G. Kar Hospital and Medical College, Tata Memorial Hospital, Bombay, Thakurpukur Cancer Hospital, the like. Some of the patients are doing well for so many years now. I feel privileged that I got the opportunity to be able to serve the humanity on such a platform.

We have primarily focused on those patients who are suffering from cancer in liver, lung, stomach, gall bladder and pancreas. These types of cancer are Chemotherapy-resistant and the possibility of regression of the disease in patients is nearly zero. If one goes through books by Hutchinson and DeVita and others on cancer, one would realise where the conventional cancer treatment is at this point of time and how much we have come ahead of them. We have treated more of those patients that lie below the 50 mark in the Karnofsky scale. A large section of patients also come from poor socio-economic background. We are not claiming for anything here but simply want to present certain important facts in front of the scientific community and the general public to make them aware and also to show the true current scenario and the inevitable future of cancer not only in India but also the whole world is about to witness. I want to show that malignant tumour can be totally regressed. The scientific community has its own ways of investigating the truth and this is called the 'Acid-test' by many. I have passed through 'Agni Pariksha' or 'Acid-test' number of times in my career and emerged victorious. My research and the cancer cases treated by me have been acknowledged all around the world by experts in different institutions, journals and publications. My research and the drug have progressed over time and got more developed in the past few years as well. I can say without any hesitation that we have prepared a drug sitting in India, that the outside world is applauding now. This treatment is not an alternative. There is nothing called alternative in this world. That what is stated to be alternative is actually a misnomer. Psorinum Therapy had shown us the end of cancer from the beginning. The position that Psorinum Therapy has gained now, if it ever gets displaced from its current position, then we have to accept that the way to detect the truth by the scientific community has too failed. I do not take pride in saying this but this is the truth. I am holding my long 35 years of struggle in front of the people and now it is up to them to decide.

Irrespective of all this, a large section of cancer patients are still not seeking my help either at the first instance or when big reputed hospitals have already showed them the door. Some of them come after undergoing Chemotherapy and Radiotherapy. People who think of themselves more knowledgeable try to do one thing and many at one go. This is where the difference lies between an educated and an illiterate poor, wherein the latter would prefer to go by my advice. The issues that I have to face treating the patients day-in and day-out are very hard to explain. The scientific

world has acknowledged the fact that the patients suffering from lung, liver, stomach, gall-bladder and pancreatic cancer are very difficult to treat and do not live for long. In most of the cases surgery is not an appropriate option, and even if it is performed encouraging results are not obtained. Even chemotherapy cannot be administered in most of the cases. Patients and their families come to us from different parts of India with the hope that we would be able to relieve them from the pain and grief. Majority of the cases are related to advanced pancreatic cancer, on whom somehow stenting has been administered, and the patients are in severe pain. At such stage a patient is normally brought to us. The most unfortunate thing is in such situations that we do not have much to do in hand. At the most we can try to give the patient the best service possible and relieve some pain. We often have to struggle hard to explain to the patient's family why chemotherapy cannot be administered. The general question we face is for how many days the patient will survive. To that we say that nobody knows after 48 hours what will happen. They ask, why. We say that a person does not die due to cancer alone. Heart failure, kidney failure, cerebral attack (CBA) and there are many other conditions that can take a life. The most frequent question is how much we can help. We say that we deal with the subject perhaps better because we have a non-toxic medicine that works provenly well in terminal condition. Along with it we have a supportive management staff that is available 24X7. We will try utmost to help. A section of patients, as a result, turn up to be treated under me.

The tale of a Toddler regarding his obscure journey ends here. Now I am in the midst of a deep study how scientific research of treatment of Cancer has developed, what are its limitations, what are its successes and failures? I had many queries which are not yet answered. I wish to know all of them. During my past days, more than once I had to be involved in confrontation with a group of Oncologists and at the same time I got the support from other Oncologists too. On the whole I tried to avoid bitter internecine fight amongst the medical practitioners and my goal was only to save the life of every individual affected with cancer and to defer their death as far as practicable, if not fully cure them from deadly Cancer.

Cancer Control is an area in which we need participation from all sections of the society. Our idea here is not to make people scared but to highlight the fact that there are areas where various agencies can put in their respective contribution. Keeping certain targets will help to monitor the programme as well as to identify the usefulness of the strategies. India can very much take up programmes and demonstrate to the World that Cancer Control is in reality feasible, and can become a model for Cancer Control Programmes in low resource settings.

Instead of being scared or evasive from the actual scenario, we,

at the CCMRCC, have been striving for the solution from a whole new perspective and from a different point of view. As has been written by Kaviguru Rabindranath Tagore:

New Deliverer –

The new age eagerly looks
To the path of your coming.
What message have you brought
To the world?
In the mortal arena
What seat has been prepared for you?
What new form of address
Have you brought to be used
In the worship of God in Man?
What song of heaven
Have you heard before coming?
What great weapon for the fighting of evil
Have you placed in the quiver....
A message of reassurance –
They seem to be promised
Deliverance, light, dawn.

(Translated by William Radice in "Rabindranath Tagore – Selected Poems": Penguin)

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CANCER – GLOBAL AND INDIAN SCENARIO

In 1937, when as a result of national policy, National Cancer Institute was set up in USA to effectively combat rising cancer incidences, there was hardly any such phenomenon in India. May be due to lack of proper diagnostic many cases remained untraced, but still it was not so alarming in India compared to the global scenario, at least till 1970s. The global scenario is more gloomy and alarming. No world war can wipe out the whole mankind or even half of the world population, but as it appears now, cancer alone can eliminate most of the world population, if its spread is not effectively regressed. Till the end of the last decade, cancer remained the second most common disease in India for maximum casualties with about 0.3 million deaths every year. The present scenario is so grave that amongst about 7.5 billion world population in 2020, about 15 million new cancer cases are expected to be diagnosed, with deaths of about 12 million cancer patients. The cancer-incidence in India has been projected to be around 2.5 million, with more or less 8 lakh new cases and 5.5 lakh deaths every year. A well-accepted survey report published by ICMR, 2009, reveals that in India total number of cancer-patients in 2004 were about 8,19,354 which increased continuously up to 9,62,832 in 2009 and 9,79,786 in 2010. By 2020, the figure is likely to cross nearly 11 lakhs. All types of cancer incidence have been found amongst the Indian population, including the cancers of lungs, breast, skin, rectum, stomach, prostate, liver, cervix, oesophagus, bladder, blood, mouth etc. causes of which may be attributed to internal factors like genetic mutations, hormonal, poor immunity as well as to external or environmental factors like life-style, food habits, industrialisation, cooking dynamics, random use of pesticides/ insecticides etc. By 2001, mainly due to environmental pollution, lung cancer became almost epidemic causing more deaths than those caused by colorectal, breast and prostate. A survey covering five Metropolitan cities in India, namely, Delhi, Mumbai, Chennai, Bangalore (now Bengaluru) and Bhopal in 1998 showed that Delhi had the maximum cancer-cases, while Bhopal had the lowest despite the infamous gas tragedy, and the data collected by the National Cancer Registry Programme of the Indian Council of Medical Research showed varying degrees of incidence of cancer in different areas of the country. Besides the most common cases of lung, breast, stomach, gall-bladder, cervical and oral cancer, other sharply rising cases of oesophageal, colorectal, Head and Neck Neoplasia (HNN) cancer are day-by-day becoming phenomenal in India. It may not be wrong to say that by now India has recorded perhaps the largest reported cases of HNN in women, as, nearly 0.2 million HNN cancer cases are diagnosed in India annually, while about 4.5 million cases worldwide.

State-wise cancer phenomenon in India is again very much interesting and leads to presume a definite link more to external factors than the internal factors as mentioned above for such varying incidences. Lung cancer cases are found in almost all states in India, while it was marginal till the end of the last century. The most affected states are Andhra Pradesh, Delhi, Himachal Pradesh, Jammu & Kashmir, Jharkhand, Kerala, Maharashtra, Manipur, Mizoram, Tripura, Rajasthan, Telangana, Uttarakhand and West Bengal. Head and Neck Neoplasia or HNN is another major form of cancer in most of the Indian states, which normally accounted

for about 25% and 10% respectively in males and females till 2010, as observed. Breast cancer incidences are quite phenomenal in almost all states of India, having registered about 10% increase during 1985 – 2000; till 2010, as many as 1 lakh new patients were detected every year, most of which used to be from rural or semi-urban areas. Stomach cancer incidences are more frequent in the North-eastern states including Sikkim, Jammu & Kashmir, of which Mizoram occupies 1st position in India and 5th position globally. Surprisingly, stomach cancer cases are also phenomenal in the non-hill states of India, like Andhra Pradesh (undivided), Goa and Tamil Nadu particularly in the tribes-dominated areas. Similarly, Gall Bladder cancer is rated as the most common abdominal malignancy in the Northern states of India and particularly along the Gange's Delta. Nearly 0.4% of all patients with Gall Bladder stones are diagnosed as having Gall Bladder cancer in the beginning of the present Century. Oral cancer including Tongue cancer is detected as the 4th common type of cancer incidences amongst males in India after lung, stomach and liver, while it is the 5th common type of cancer in the females after Cervical, breast, stomach and lung. Interestingly, most affected states are again in the tribal areas of the country, including Assam, Nagaland, Mizoram, Manipur, Arunachal Pradesh, Meghalaya, Sikkim, Bihar, Odisha, Madhya Pradesh, Andhra Pradesh, Goa, Chattisgarh, Jharkhand and Himachal Pradesh, mainly because of Tobacco abuses. There is hardly any doubt that cervical cancer is another very common killer malignancy in India. While it has consistently risen in developing countries during last few decades, the number of cervical cancer incidences in women in India has gradually decreased during the first decade of the present Century, may be due to increasing awareness and precautionary measures adopted by them. Incidences of oesophageal cancer in India is moderately high, due to diets and lifestyles, as observed by the Researchers, and are found mostly in the states of Assam, Jammu & Kashmir and Karnataka. While some incidences of oesophageal cancer have been noticed in the states of Meghalaya and Haryana, a good number of cases of Non-Hodgkin's lymphoma have been detected in the states of Chattisgarh and Uttarakhand. Incidences of ovary, prostate and brain cancer are quite high in the state of Rajasthan compared to other states, the reports say. It may also be kept in mind that in India cancer incidences are generally under-reported or under-stated, in particular from the rural areas, mainly due to lack of diagnostic infrastructure and technique. In many cases, the cancer incidences get unreported because of suppression or non-reporting of such cases. Depending on the sub-site of origin of cancer and its clinical extent it was observed in 2005 that a 5-year survival rate of the disease normally varied from 20 – 90% in India.

Causes of cancer are no longer limited to genetic or biological factors like inherited mutations, hormones or immunities in the body. External or environmental or inhabitational factors like diet, food habits, consumption of tobacco and spurious liquor or other intoxicants, radiation, air and water pollutions, random use of pesticides in production of food items, use of preservative chemicals or agents in food-packaging, random use of various types of repellants, drug-abuses etc. are now more responsible for such epidemic-like spread of cancer. A significant cause of cancer has now been observed to be in the life-style and food. Extensive surveys in various hospitals and key areas of the country during 2001 – 2010 have revealed that improper or imbalanced diet is one of main causes of growing cancer

incidences, so much that about 70% of colorectal cancer cases are attributed to this factor. Charcoal cooking of red meat, smoked beef, pork or other red meats, non-use of turmeric in cooking of any vegetable or non-vegetarian item, improper use of various chemical agents while cooking, keeping food-items in plastic containers, over-use of preservatives etc. are observed to have been contributing to cancer incidences to a very large extent. Food kept in plastic containers and heating of cooked food items in plastic bowls give rise to serious risks of breast and prostate cancers, the surveys report. Although a section of researchers have observed that cooking of food by most of the Indians in very high cooking-temperature or pre-heating of food for long duration before taking meal generally lead to high risks stomach, mouth, pharyngeal, oesophageal cancers, still in the North-eastern states of India where large sections of tribal are in the age-old habit of consuming half-boiled or semi-boiled vegetables, leafs and lentils are now detected with stomach and other types of cancer mainly due to rampant spray of pesticides or like-chemicals in such vegetable plants. It is by now well-accepted that vegetarianism practised by a large section of Indians is associated with much lower risks of prostate, oesophageal, oral and breast cancers. Beans, chickpeas, lentils and pulses, if produced through organic farming, significantly arrest the various types of cancer incidences, as has been scientifically observed. In 1997, the World Cancer Research Fund got various surveys conducted relating to cancer-scenario in India, and found that the Indian diet containing adequate quantities of vegetables, fruits, and fibre-rich grains provide an effective protection against growing risk of colon and breast cancers. Similarly, the oriental researchers have observed that the traditional cooking procedure in India with balanced use of turmeric, ginger and cumin, and also cooking at a certain boiling-point often reduce the risks of various types of cancer. Because of growing demands, farmers are being compelled to produce more vegetables and other farm-produces for which they are resorting to the new concept of hybrid production and to rampant use of chemical fertilizers and pesticides. Traces of sprayed pesticides on the leaves or stem of the vegetables and other eatables continue to remain unabsorbed or unneutralised, and are being regularly transported to human body through the intakes of such food and water, to the effect of spoiling the character and fabric of our genes and hormones. This associated with consumption of tobacco in all forms, smoking and reverse-smoking, drug-abuses, excessive use of insecticides and repellents at home, indiscriminate use of cheap body-deodorants and room-fresheners, pollution of air and water at all levels, and frequent exposure to radio-active agencies or environment are the latest causes of spiral growth of cancer. It is stated that about 10% cancer incidences are due to radiation effect and the phenomenal rise of lung cancer and other associated types of cancer cases in Jharkhand, Meghalaya, Odisha, Maharashtra and Rajasthan are attributed to this factor alone, while about 90% of cancer cases are caused by environmental pollutants. Again low socio-economic conditions, poor diet, infections of viral origin etc. are also responsible for various types of cancers amongst the economically weaker section of the people. As in India, cancer incidences are highly under-reported, so it may not be practical to compare the Indian scenario with the global scenario with any particular emphasis.

On the basis of GDP, India stands at the 11th position in the world around during the end of the last decade, but like the health of its population its economy is also being

alarmingly affected by epidemical spread of cancer. Expenditure on cancer-patients includes both direct and indirect expenses. While direct expenses include costs of expensive medicines, hospitalisation, expensive pathological and radiological tests, doctors' consultancy fees, cost of expensive chemotherapy/ radiation, costs of travel, lodging etc., indirect losses include loss of income of the patient's family during treatment as well as due to premature death of the patient if he or she is the earning member of the family, destitution of the family in case of the patient's death, and irreparable damage to the future of the dependent children as well as the business-house if such patient is self-employed or entrepreneurs. There are other resultant impact of sudden cancer-deaths or prolonged cancer-treatment on the patient's family, which varies in degree or ratio on the basis of age of such patient and dependence of the family. On the basis of data available till 2004, a calculation in terms of money, of the impact of cancer on the Indian economy was prepared which shows as follows:

Year(1)	No. Of Cancer patients in India(2)	Total Expense in U. S. Dollar (million)(3)
2004	819354	215.16
2005	846635	224.11
2006	863575	233.11
2007	907838	245.30
2008	846172	232.89
2009	962832	274.10
2010	979786	270.06

[Source: Imran Ali, Waseem A. Wani and Kishwar Saleem: Cancer Scenario in India with Future Perspectives, Cancer Therapy, Vol. 8, pp. 56 – 70, 2011]

Such impacts are most felt on the economically weaker section of the people compared to the rich; more phenomenal in the rural or semi-urban areas compared to urban areas; more devastating in the male-dependant farming sector than in other tertiary sector; more ruinous in respect of micro or nuclear families compared to macro or joint families. In case of massive population-wash due to cancer, the worst victim will be the economically weaker section in India, which comprises nearly 30 – 40% of the total population of India. Although relatively cheaper than in the west, cancer treatment is still unaffordable for poor and middle-class Indians who do not have even the health insurance policies. In a nut-shell, I would like to present the prevailing health scenario in India here.

With scientific research I here bring along economics, sociology and all other aspects that I had no knowledge about earlier, but in my journey I have come across many of such.

As of March 2016, the population of India is 1.32 billion people, among which the

current male population is 681 million (51.6%) and current female population is 638 million (48.4%). The figures show that India represents almost 17.85% of the world's population, which means one out of six people on this planet live in India. Although, the crown of the world's most populous country is on China's head for decades, India is all set to take the numero uno position by 2030. With the population growth rate at 1.2%, India is predicted to have more than 1.53 billion people by the end of 2030.

More than 50% of India's current population is below the age of 25 and over 65% below the age of 35. About 72.2% of the population lives in some 638,000 villages and the rest 27.8% in about 5,480 towns and urban agglomerations. The birth rate (child births per 1,000 people per year) is 22.22 births/1,000 population (2009 est.) while death rate (deaths per 1000 individuals per year) is 6.4 deaths/1,000 population. India has the largest illiterate population in the world. The literacy rate of India as per 2011 Population Census is 74.04%, with male literacy rate at 82.14% and female at 65.46%. Kerala has the highest literacy rate at 93.9%, Lakshadweep (92.3%) is on the second position and Mizoram (91.6%) is on third.

Some of the reasons for India's rapidly growing population are poverty, illiteracy, high fertility rate, rapid decline in death rates or mortality rates and immigration from neighbouring countries.

The current rural and urban population are 833 million and 377 million respectively and roughly 30 per cent of India's population still lives below the poverty line (India in Figures 2015, www.mospi.com) as in the year 2011.

In the following years the Indian population forecast is as follows:

Year	Population (in billions)	Yearly % change	Country's share of World Population in %	World Population(in billions)
2020	1.35	1.08	17.54	7.72
2025	1.42	0.95	17.55	8.08
2030	1.48	0.80	17.52	8.42
2035	1.53	0.66	17.45	8.74
2040	1.57	0.52	17.32	9.08
2045	1.60	0.40	17.16	9.31
2050	1.62	0.29	16.96	9.56

The press note released by The Planning Commission, July 2013, aims to review the methodology for estimation of poverty for 2011-12 under the Chairmanship of Prof. Suresh D. Tendulkar. The following two tables will show:

1. the estimates of state wise poverty lines for rural and urban areas for 2011-12.
2. The percentage and number of persons below poverty line for all States/UTs for rural areas, urban areas and combined.

The all India poverty ratio is obtained as state-population weighted average poverty ratio, and the all India poverty line is the per capita per month expenditure that corresponds to the all India poverty ratio.

For 2011-12, for rural areas the national poverty line using the Tendulkar methodology is estimated at Rs. 816 per capita per month and Rs. 1,000 per capita per month in urban areas. Thus, for a family of five, the all India poverty line in terms of consumption expenditure would amount to about Rs. 4,080 per month in rural areas and Rs. 5,000 per month in urban areas. These poverty lines would vary from State to State because of inter-state price differentials.

State specific Poverty Lines for 2011-12

S.No.	States	Monthly per capita(Rs.)	
		RURAL	URBAN
1	Andhra Pradesh	860	1009
2	Arunachal Pradesh	930	1060
3	Assam	828	1008
4	Bihar	778	923
5	Chhattisgarh	738	849
6	Delhi	1145	1134
7	Goa	1090	1134
8	Gujarat	932	1152
9	Haryana	1015	1169
10	Himachal Pradesh	913	1064
11	Jammu & Kashmir	891	988
12	Jharkhand	748	974
13	Karnataka	902	1089
14	Kerala	1018	987
15	Madhya Pradesh	771	897
16	Maharashtra	967	1126
17	Manipur	1118	1170
18	Meghalaya	888	1154
19	Mizoram	1066	1155
20	Nagaland	1270	1302
21	Odisha	695	861
22	Punjab	1054	1155
23	Rajasthan	905	1002
24	Sikkim	930	1226
25	Tamil Nadu	880	937
26	Tripura	798	920
27	Uttarakhand	880	1082
28	Uttar Pradesh	768	941
29	West Bengal	783	981
30	Puducherry	1301	1309
	All India	816	1000

Note: Computed as per Tendulkar method on Mixed Reference Period (MRP)

**Number and Percentage of Population below
poverty line by states - 2011-12**

(Tendulkar Methodology)

S.No.	States	Rural		Urban		Total	
		%age of Persons	No. of Persons (lakhs)	%age of Persons	No. of Persons (lakhs)	%age of Persons	No. of Persons (lakhs)
1	Andhra Pradesh	10.96	61.80	5.81	16.98	9.20	78.78
2	Arunachal Pradesh	38.93	4.25	20.33	0.66	34.67	4.91
3	Assam	33.89	92.06	20.49	9.21	31.98	101.27
4	Bihar	34.06	320.40	31.23	37.75	33.74	358.15
5	Chhattisgarh	44.16	88.90	24.75	15.22	39.93	104.11
6	Delhi	12.92	0.50	9.84	16.46	9.91	16.96
7	Goa	6.81	0.37	4.09	0.38	5.09	0.75
8	Gujarat	21.54	75.35	10.14	26.88	16.63	102.23
9	Haryana	11.64	19.42	10.28	9.41	11.16	28.83
10	Himachal Pradesh	8.48	5.29	4.33	0.30	8.06	5.59
11	Jammu & Kashmir	11.54	10.73	7.20	2.53	10.35	13.27
12	Jharkhand	40.84	104.09	24.83	20.24	36.96	124.33
13	Karnataka	24.53	92.80	15.25	36.96	20.91	129.76
14	Kerala	9.14	15.48	4.97	8.46	7.05	23.95
15	Madhya Pradesh	35.74	190.95	21.00	43.10	31.65	234.06
16	Maharashtra	24.22	150.56	9.12	47.36	17.35	197.92
17	Manipur	38.80	7.45	32.59	2.78	36.89	10.22
18	Meghalaya	12.53	3.04	9.26	0.57	11.87	3.61
19	Mizoram	35.43	1.91	6.36	0.37	20.40	2.27
20	Nagaland	19.93	2.76	16.48	1.00	18.88	3.76
21	Odisha	35.69	126.14	17.29	12.39	32.59	138.53
22	Punjab	7.66	13.35	9.24	9.82	8.26	23.18
23	Rajasthan	16.15	84.19	10.69	18.73	14.71	102.92
24	Sikkim	9.85	0.45	3.66	0.06	8.19	0.51
25	Tamil Nadu	15.83	59.23	6.54	23.40	11.28	82.63
26	Tripura	16.53	4.49	7.72	0.75	14.05	5.24
27	Uttarakhand	11.62	8.25	10.48	3.35	11.26	11.60
28	Uttar Pradesh	30.40	479.35	26.06	118.84	29.43	598.19
29	West Bengal	22.52	141.14	14.66	43.83	19.98	184.998

30	Puducherry	17.06	0.69	6.30	0.55	9.69	1.24
31	Andaman & Nicobar Islands	1.57	0.04	0.00	0.00	1.00	0.04
32	Chandigarh	1.64	0.004	22.31	2.34	21.81	2.35
33	Dadra & Nagar Haveli	62.59	1.15	15.58	0.28	39.31	1.43
34	Daman & Diu	0.00	0.00	12.62	0.26	9.86	0.26
35	Lakshadweep	0.00	0.00	3.44	0.02	2.77	0.02
	All India	25.70	2166.58	13.70	531.25	21.92	2697.83

- Notes :
1. Population as on 1st March 2012 has been used for estimating number of persons below poverty line. (2011 Census population extrapolated)
 2. Poverty line of Tamil Nadu has been used for Andaman and Nicobar Island.
 3. Urban Poverty Line of Punjab has been used for both rural and urban areas of Chandigarh.
 4. Poverty Line of Maharashtra has been used for Dadra & Nagar Haveli.
 5. Poverty line of Goa has been used for Daman & Diu.
 6. Poverty Line of Kerala has been used for Lakshadweep.

The percentage of persons below the Poverty Line in 2011-12 has been estimated as 25.7% in rural areas, 13.7% in urban areas and 21.9% for the country as a whole. The respective ratios for the rural and urban areas were 41.8% and 25.7% and 37.2% for the country as a whole in 2004-05. It was 50.1% in rural areas, 31.8% in urban areas and 45.3% for the country as a whole in 1993-94. In 2011-12, India had 270 million persons below the Tendulkar Poverty Line as compared to 407 million in 2004-05, that is a reduction of 137 million persons over the seven year period.

During the 11-year period 1993-94 to 2004-05, the average decline in the poverty ratio was 0.74 percentage points per year. It accelerated to 2.18 percentage points per year during the 7-year period 2004-05 to 2011-12. Therefore, it can be concluded that the rate of decline in the poverty ratio during the most recent 7-year period 2004-05 to 2011-12 was about three times of that experienced in the 11-year period 1993-94 to 2004-05.

It is important to note that although the trend decline documented above is based on the Tendulkar poverty line, an increase in the poverty line will not alter the fact of a decline. While the absolute levels of poverty would be higher, the rate of decline would be similar.

Keeping this in the backdrop while talking about health system in India, the physician density is 0.65 physicians/1,000 population (2009) i.e., India has just one doctor for every 1700 people. There are about 6 – 6.5 lakh doctors available, but India would need 4 lakh more by 2020 to maintain the required ratio of one doctor per 1,000 people, with an additional force of 12 lakh nurses.

We here start talking from the perspective of rural economy since according to the provisional data released by Census India, the trends of Urban and Rural Population of India

shows that the rural population is 68.84% compared to the urban population of 31.16%. Also the urban areas have seen a much higher growth rate as compared to rural areas.

If shortage of doctors is one problem, their unwillingness to work in the rural hinterland is another.

“Despite the efforts of the government and incentives offered, medical students or doctors are not showing interest in working in rural areas. I wonder what is wrong with the doctor fraternity,” said Gulam Nabi Azad (Minister of Health & Family Welfare, 2009–14), implying that it’s no longer a “service” but a “profession”.

The reasons of rural reluctance are due to shortage of doctors, their unwillingness to work in the rural hinterland and creation of artificial scarcity in the area and high concentration.

But the doctors are not to blame either. Surveys of rural healthcare facilities have revealed poor infrastructure, non-availability of medicines, equipment and even lack of basic necessary infrastructure. Also shortage of human resources is a distressing feature of India’s healthcare services.

In rural areas and semi-urban areas of the state, there is scarcity of doctors and specialists besides an acute shortage of well-trained paramedical staff, including nurses. Despite the success of the National Rural Health Mission (NRHM), the shortage of human resources is a distressing feature of India’s healthcare services. Even the Planning Commission has conceded that availability of healthcare services is quantitatively inadequate.

There are 387 medical colleges in the country – 181 in government and 206 in private sector. India produces 30,000 doctors, 18,000 specialists, 30,000 AYUSH graduates, 54,000 nurses, 15,000 Auxiliary Nurse Midwifery (ANMs) and 36,000 pharmacists annually.

Many doctors posted in the hospitals in the rural areas remain absent for long periods. And in the absence of doctors, patients visiting the healthcare centres are treated by stand-ins—pharmacists and even nurses.

The situation in the medical college hospitals is equally grim.

At the start of 11th Plan, the number of doctors per lakh population in the country was only 45, whereas the desirable number is 85 per lakh population. As per rural health statistics 2012, there were 1,48,366 sub-centres, 24,049 Primary Health Centres (PHCs) and 4,833 Community Health Centres (CHCs) functioning in the country.

Compared to requirement for existing infrastructure, there was a shortfall of 74.9 per cent of surgeons, 65.1 per cent of obstetricians and gynaecologists, 79.6 per cent of physicians and 79.8 per cent of paediatricians. Overall, there was a shortfall of 69.7 per cent specialists at the CHCs.

Talking about the budget allocation for healthcare in India, the total plan expenditure incurred by the Union Government for public health for the years 2007-08 to 2010-11 is as follows:

Year	Allocation (Rs. In Crores)	Expenditure (Rs. in Crores)
2007-08	15291	12947.47
2008-09	16543	15130.08
2009-10	19534	17636.49
2010-11 (up to 28.02.2011)	22300	17589.84
2011-12	26760	

[Government has increased the plan allocation for the public health spending to Rs. 26,760 crore in 2011-12 from Rs. 22,300 crore in 2010-11 and Rs.19,534 crore in 2009-10 respectively. This information was given by Union Minister of Health & Family Welfare Shri Ghulam Nabi Azad in written reply to a question in the Lok Sabha.]

The 2015-16 budget neither showed any significant ramp up in public funding for healthcare, nor did it provide any impetus to research and development in the pharmaceutical and life sciences. It may have kick-started thinking on the healthcare agenda, but the latter needs far more attention to be able to make a significant impact.

While there is an intent within the government for an integrated healthcare system to ensure affordable and accessible healthcare for all, it is not reflected in the Budget allocation to healthcare for 2015-16, which was marginally higher than last year. The opening of six new large public hospitals across the country and the introduction of better health insurance options are strong symbols of change. However, it is unfortunate that the government missed the opportunity to provide a strong impetus to the pharma and life-sciences sector. The budget also stopped short of prescribing an investment formula for universal healthcare.

The National Health Policy 2015 draft prepared by the ministry of health and family welfare rues the low priority accorded to healthcare by successive governments. “It is unrealistic to expect to achieve key goals in a Five-Year Plan on half the estimated and sanctioned budget,” the report says, adding, “The failure to attain minimum levels of public health expenditure remains the single-most important constraint.”

India urgently needs to raise public health spending to at least 2.5% of GDP if the government’s promise of universal health coverage is to be fulfilled. Without this, it will not be able to make much progress towards meeting the healthcare needs of 1.25 billion people. The Indian pharma industry has expressed its willingness to partner the government to help it deliver on the agenda of ‘Health for All’ and enable affordable access to medicines and diagnostics that meet the needs of all patients.

The Indian pharma sector has already done the country proud by being the largest supplier of generic drugs to the world. But, unfortunately, it has missed out on addressing India’s healthcare challenge due to the absence of strong political will and the low priority accorded to this sector.

The industry’s expectations were belied when Budget 2015 did not lend support to its capital investment needs, didn’t incentivise R&D investments or provide any significant tax exemptions for innovation. Other than announcing the Atal Innovation Mission, this Budget has done precious little to enable the culture of ‘Innovate in India’, or to encourage companies

that focus on innovation. It did not even reverse the service tax imposed on clinical research organisations introduced last year.

The government needs to seriously invest in 'Swastha India' by revitalising existing public health infrastructure, creating new medical centres, promoting partnership models, enabling indigenous manufacturing and incentivising research and development.

Although our data does not suggest, still it is very much visible that there exists severe under allocation of resources in the health sector as well as under utilization of the allocated.

As, I had stated earlier, in India the doctor-to-patient ratio is skewed. Even as cancer is fast turning into an epidemic in India, with about two million registered patients, the country is facing an acute shortage of oncologists, surgical oncologists and radio-therapists. Official data show there are only about 1,000 trained oncologists in the country and the ratio of oncologists to cancer patients is about 1:2,000 (as of 2012).

- **1.8 million** cancer patients in India (within five years of diagnosis)
- **683,000** deaths due to cancer in 2012
- **Over 1 million** new cancer incidents getting added every year.
- **1,000** trained oncologists in the country (doctor-patient ratio of 1:2,000)
- **27** Dedicated cancer hospitals in India

Indians are at high risk of acquiring cancers due to high rates of smoking, tobacco use, occupational risks, and unhygienic residential living conditions. The prevalence of cancer in India is affecting the economy of the country. Indian economy has been affected by the alarming rise of cancers in the last decade.

The disease has wiped out entire life savings and even forced some people to sell their homes. Although relatively cheaper than in the West, cancer treatment is still unaffordable for poor and middleclass Indians, who often do not have health insurance.

While the incidence of cancer is rising, insurance is a big stumbling block. Only a small fraction of Indians has health insurance and those who do rarely have critical illness cover. Worse, the insurance cover that people choose is often so low that it is meaningless for diseases such as cancer. Of the 40 million individual policies sold in 2011, the average amount per policy was just Rs 1.9 lakh. A higher Rs 10-lakh policy would mean a Rs 17,000 to Rs 50,000 annual premium, depending on the age of the policyholder.

Why is cancer treatment so forbiddingly expensive? The main reason is the staggeringly high cost of equipment and setting up a cancer hospital: doctors estimate a 100-bed cancer speciality hospital would need an investment of up to Rs 100 crore, excluding the cost of land in most cases.

Hospitals have to pay a fortune for imported equipment. For example, a linear accelerator, which is used for radiation therapy, costs around Rs 10 crore, plus an import duty of around Rs 1 crore. Similarly, a PET CT scan machine, used to pinpoint the location of cancers, is anywhere between Rs 3 crore and Rs 6 crore. The costliest is a Cyber Knife used for radiotherapy which costs around Rs 30 crore. Apart from equipment, cancer treatment medicines are expensive.

Some breast cancer patients, for example, need targeted treatment drugs, such as Herceptin or Herclon, made by global major Roche, which cost around Rs 75,000 for a course; a patient could need up to 17 courses. Similarly, a drug called Avastin - used to treat colon, kidney, lung and gall bladder cancer - can add around Rs 8 lakh to a patient's bill at around Rs 1 lakh a cycle. Though companies have patient support programmes, most middle-class patients end up paying 70 to 80 per cent of the drug cost.

With such high costs, cancer treatment is often out of the reach of millions. Take the case of a 16-year-old girl being treated for a brain tumour at Hyderabad's Apollo Hospital. Her parents - a teacher and a government employee - plan to dip into savings set aside for her education and marriage to pay for her treatment, which is expected to cost around Rs 8 lakh. "She wants to be a doctor and we have been saving for that. But what good are the savings if we are not able to save her," says her mother.

Doctors are concerned at the growing number of cancer cases in India. Although there is no nationwide data, estimates extrapolated from the Indian Council of Medical Research's cancer registry programme in some areas indicate there are about one million new cancer cases each year. A Planning Commission report estimates that about 2.8 million people have cancer at any point of time and half a million die of the disease each year. The number of cases is expected to go up because of an increase in life expectancy, changing lifestyles and risk factors such as high tobacco use.

Let one visit any cancer hospital in the country and one will find that there are dozens of people starting from little children to the elderly dealing with the trauma of the disease.

One way to bring down the crippling costs is through compulsory licensing of cancer drugs, which allows drug companies to make generic versions of patented medicines at cheaper prices.

Today patients also have a lot more hospitals to choose from. Fifteen years ago, Tata Memorial Hospital was among the few cancer treatment facilities in India. Cancer diagnostics and treatment facilities such as PET scans were limited. Today, the country has about 80 cancer hospitals, and several chains.

Cancer hospitals are mostly concentrated in big cities, though some chains, such as Bangalore's Healthcare Global, have set up hospitals in small towns. Several charitable hospitals also help poor get cancer treatment. But the question here is how many people are aware of it and has access to them.

A new study on access to healthcare facilities shows that availability of healthcare services is skewed towards urban centers with these residents, who make up only 28% of the country's population, enjoying access to 66% of India's available hospital beds, while the remaining 72%, who live in rural areas, have access to just one-third of the beds.

Insufficiencies in public healthcare services have driven people across socio-economic strata to private healthcare facilities leading to issues of affordability challenges. In 2012, 61% of rural patients and 69% of urban patients chose private in-patient service providers, up from 40% reported in a 1986-87 government survey.

But since the cost of treatment at private healthcare facilities is at least 2 to 9 times

higher than at public facilities, it leads to the factor. Poor patients receiving outpatient care for chronic conditions at a private facility spent on an average 44% of their monthly household expenditure per treatment, against 23% for those using a public facility, says the study conducted by IMS Institute for Healthcare Informatics.

According to the IMS study, the lack of accessible healthcare facilities in rural areas, the difficulty in accessing transport and the loss of earnings means patients postpone treatment, or make do with facilities that may be closer but are not cost-effective or even suited to their needs.

The study, which was based on a survey of nearly 15,000 households across 12 states, says that a 40-45% reduction in out-of-pocket expenditures for both outpatient and inpatient treatments can be achieved by addressing physical accessibility of healthcare facilities, availability and capacity of needed resources; quality and functionality of service, and affordability of treatment relative to a patient's income.

Now-a-days there are also many NGOs that help patients and their families deal with the emotional trauma of cancer.

The major reason for cancer deaths in India is the high treatment cost and inferior quality of equipments available in nearly 40% of cancer centres in India. So best solution to avoid expensive treatments is early detection and adopting preventive measures and lifestyle changes. Modern technology has led to wonderful cures but also costs good money.

Talking about the poor, poor patients being given excellent and more importantly subsidized cancer treatment at one of the leading government hospitals in Chennai. However, the same thing cannot be said regarding all the government hospitals across the country. Apart from these hospitals and charitable organizations poor cancer patients have no other option for treatment in India. Some State Governments do offer insurance coverage even to the poor but it is unlikely that so schemes will suffice for cancer treatment. Unfortunately, the Central Government does not seem to have any strategy to deal with the rising cases of cancer treatment. At least for TB and HIV, the central government has some kind of public funded programs in place such as NACO etc. Cancer unfortunately has simply dropped off the list.

Again, cancer cannot be diagnosed that accurately by a single test. The cancer diagnosis starts with complete evaluation of a patient. The evaluation includes history taking and physical examination along with diagnostic testing. Many laboratory and imaging tests are needed to detect and determine cancer.

Cancer diagnosis is helpful in confirming or eliminating the presence of disease, monitoring the progress of the disease and to plan for effective treatment. Diagnostic procedures for cancer may include imaging, laboratory tests, tests for tumour markers, tumour biopsy, endoscopic examination, surgery, or genetic testing.

Also cancer is indicated by X-ray, CT Scan, MRI, PET Scan, Nucleus Scan and a few blood tests like CA125, PSA, AFP, LDH etc.. But ultimately FNAC, cytology, biopsy is conducted. Without this proof we cannot document a cancer to be official.

Besides our limited resources, number of available Onco-pathologists, Onco-surgeons, radiotherapists etc., their number is very small. The number that we have in hand, and with

our current economic status and budget allocation towards health particularly cancer, it is not possible to detect cancer for more than 15 crore people.

So 115 crore people out of 130 crore are lying beyond the reach. As we have said that other diseases has one or two types but cancer has 200, among them many are resistant to radiotherapy and chemotherapy. Cancer does not distinguish between rich and poor.

By 2025 it is being said that cancer would multiply five times. Among them a large population would lie in the age group of 30-50. Children are dependent on their parents, many people on their spouse, many aged parents on their adult children. If such a person dies the entire family falls into serious psychological and financial problems. Their rehabilitation, financial and social security, specially the ladies, should be looked after. In the Hindu community a large number of people do not remarry after their spouse dies because of which one has to bear the responsibility and the entire financial burden upon his/her shoulders. We need to keep this in our mind as well.

With more than 1,300 persons succumbing to cancer every day, it has become one of the major causes of death occurring in the country due to communicable and life-style ailments.

As per data of the National Cancer Registry Programme of the India Council of Medical Research (ICMR), the estimated mortality rate due to cancer saw an increase of six per cent approximately between 2012 and 2014.

“There has been close to 5 lakh deaths due to cancer in the country in 2014,” said a senior Health Ministry official.

Total of 4,91,598 people died in 2014 out of 28,20,179 cases, while in 2013 it was 4,78,180 deaths out of 29,34,314 cases reported and in 2012, around 4,65,169 people lost their lives due to the disease when the number of cases stood at 30,16,628.

“Large number of ageing population, unhealthy lifestyles, use of tobacco and tobacco products, unhealthy diets, lack of diagnostic facilities, etc. are some of the factors that can be attributed to the increase in the number of cancer deaths,” the official said.

The government has approved a scheme in 2013-14 for enhancing the specialised consultative care for cancer in the country and guidelines for strengthening of the facilities were circulated to the states in January 2014.

“The need for political commitment and action is at the heart of the solution to India’s growing cancer burden,” said Mohandas Mallath, MBBS, MD, lead author of 1 in a series of 3 papers on the cancer burden in India published in The Lancet.

“The extent to which death and illness from cancer will actually increase in the next 20 years will depend a lot on the investments made in future decades in tobacco control, healthcare delivery, cancer research, clinical trials, and increasing the public awareness as to how we can all help to reduce the risk of cancer by eliminating risky behaviours such as smoking, vaccinating those at risk of cancer causing infections and following a healthy diet and lifestyle,” Dr. Mallath said in a statement.

The average cost of treating a typical patient with cancer at a government facility runs about \$593, but the average annual income per person is only \$1219, and 27.5% of the

population live on or below \$0.4 per day, the report points out.

“Most district hospitals and even regional cancer centres do not have the facilities needed to provide quality cancer care to the people who need them,” Dr. Pramesh’ commented in a statement. “Because of the extremely low level of government and state funding for healthcare in India, a cancer diagnosis is increasingly responsible for catastrophic expenditures from the patients’ own pockets, which has the potential to negatively affect not only the individual with cancer, but also the welfare and education of several generations of their family.”

Cancer Control is an area in which we need participation from all sectors of the society. Delivering affordable and equitable care for cancer is one of India’s greatest public health challenges. Our idea here is not to make people afraid but to highlight the fact that there are areas in which various agencies can put in their contributions. Keeping targets will help to monitor the programme as well as to identify the usefulness of the strategies. India could take up programmes and demonstrate to the World that Cancer Control is feasible and become a model for Cancer Control Programmes in low resource settings.

4 criteria that makes a ideal health care system that could be suggested-

First, universal access, and access to an adequate level, and access without excessive burden.

Second, fair distribution of financial costs for access and fair distribution of burden in rationing care and capacity and a constant search for improvement to a more just system.

Third, training providers for competence, empathy and accountability, pursuit of quality care and cost effective use of the results of relevant research.

Last, special attention to vulnerable groups such children, women, disabled and the aged.

What is the real need of the time is such a therapy which is cheap, quality proven and effective, a treatment which is full of honesty, associated with homely-care and free and frank pre-treatment counselling as well as post-treatment counselling, and a proper rehabilitation policy, because victimisation of cancer in many cases is a social cause due to environmental pollution and other social factors or health-hazards. Therefore, approach to cancer-cure in India should be a total package, and I am seriously, honestly and whole-heartedly in search for the same.

CANCER :

A DISPASSIONATE OBSERVATION-A TRUTH

Asim Chatterjee

We now intend to discuss a new side of cancer. How is the diagnosis done? When the disease is ascertained, how do the patient and his/her family friends react to the truth? Cancer is generally determined by GPs i.e. general practitioners. They are approached first by a patient. The symptoms like swelling of body parts, existence of cough for a long time makes the practitioners suspect of cancer. Other symptoms include bleeding with cough and urine, choked voice, uneasiness at times of eating, repeated vomiting, ascities in stomach, breathing problem, and effusion in chest X-ray etc. Doctors suggest some investigations thereafter. It is followed by x-ray, CT scan, endoscopy, Colonoscopy and other blood tests in order to examine whether WBC counts in blood, PCA in blood and CA 125 have increased. If GPs suspect of cancer, patients are sent to Onco-Surgeons. The Onco-Surgeon then investigates in details to find out whether any part of the body needs a surgery. If needed, the particular part of the body is operated and removed to send to pathologist for further investigation. The pathologist first collects portions of the operated material to carry out various examinations, and then he cuts a thinner portion of the material and places it on a glass slide. This method is called biopsy. At times when surgery is not possible at the infected area, a needle is penetrated by special methods in order to examine the liquid of that area. This is called FNAC (Fine Needle Aspiration Cytology).

In case of effusion in lungs, water is taken out by a syringe and sent for investigation. If malignant cells are detected in that liquid then presence of cancer is ascertained. Even if cancer cells cannot be located and the liquid is red in colour then also absence of cancer cannot be ensured. Cells are examined invariably to be sure about the existence of cancer. Similarly water from stomach is also collected by a syringe and sent to pathologists. Sometimes malignant or cancer cells can be located, if not, then investigation continues to find out if cancer cells are there in any other part of the body.

Blood Cancer is suspected of when blood tests show an increase in WBC counts and a simultaneous reduction in RBC counts. Bone-marrow test helps to know if at all it is cancer. Increase of PCA level of blood is an indication of cancer. Then Sonography or CT Scan of lower abdomen examines the size of the prostate, or if a tumour is attached to the prostate. A tissue of that area is examined by Cystocopy. Ovary cancer is suspected if cancer cells can be detected in stomach ascitis and if CA-125 level increase in blood. The tumour is examined by CT scan thereafter. Sometimes glands swell. Investigation follows. Hodgkin or non-Hodgkin lymphoma is suspected. In case of severe bone pain, X-ray or whole body bone scan determines the destruction of bones and Multiple Myeloma is suspected of. It is examined by either FNAC or bone-marrow test.

A patient or his/her family becomes very upset, the moment they come to know that he/she or one of their beloved ones has been a victim of cancer. The situation is similar to

that of facing the order of being hanged till death. Just like the accused who appeals to the High Court or Supreme Court depending on financial ability or manpower, similarly cancer patients also run to collect resources according to their ability to fight the danger. People run to different centres according to their best ability. Notable centres of India include Mumbai, Tata Memorial Hospital or Vellore, Chennai Apollo or Hyderabad Apollo. Some even run to foreign centres like M. D. Anderson of USA. People with lesser financial ability run to Kolkata AMRI, Apollo etc. Others approached hospitals like Chittaranjan National Cancer Hospital, SSKM Hospital or Medical College Hospital for their treatment. People face greatest trouble who being under several influences do visit different states despite of financial weakness. Treatment of cancer except some specific types has been almost at a stagnant status since long, no matter how much we become excited with new inventions.

Different types of cancer respond to nothing except palliative management just to reduce the pain temporarily. General mass symbolizes cancer with a particular type of illness. They believe that overhead charges or expenses under renowned doctors at renowned institutions may yield some result. If the news reaches that someone is in good health for a long time under some renowned doctor in some other state then no one bothers about the type of cancer. On the other hand, an untimely unfortunate death in any West Bengal Hospitals leads to the conclusion that no one survives long in these hospitals but do survive in hospitals of other states. People run to different places at the cost of all their belongings. Perhaps at that moment — they would remember quotes of great poet Madhusudan Datta – ‘Ashar chhalane bhule ki fal lovinu hai! (How much bitter it tastes to move in other land recklessly helter skelter without any positive result.)

We should be aware that the entire Medical world is one family – so know-how about the treatment is same as in Bangladesh and as in Sri Lanka. The first thing we must remember that generally none of the cancer patients survive long but their family survive for long. As such, if all the resources of a family is spent over the dying person then obviously the family will walk towards a future of financial, physical and mental uncertainty and destruction. We carried out a study of some families of cancer patients. The study veered round the (1) Physical, mental and financial condition of a family two years before, one of whose members has been very recently victimized by cancer. (2) What was the present condition of the family during the time patient survived and treatment continued and (3) what was the condition of the family two years after the person expired. Our observation centred mostly on those of young age and those who are the main bread earners of the family. The agony and dreadfulness of our findings cannot be expressed in words. It's too difficult and tough to narrate. According to cancer specialists, 2020 will have cancer at every door. Can you imagine how awful it might be if we do not prepare ourselves in advance to face the danger?

(Source : Mukta Asar, Bengali little Magazine 4th issue, Editor - Gautam Chatterjee)

THE MANAGEMENT OF CANCER IN TOTALITY – CAN INDIA TAKE A LEAD IN THEORY AND APPLICATION?

In this repertory we, in our humble capacity have tried our best to –

- 1) Depict the modern treatment of cancer throughout the world together with its research works,
- 2) Illustrate our efforts in respect of treatment of cancer simultaneously with a non-conventional method known as Psorinum Therapy,
- 3) Elucidate a comparative study in order to establish and evaluate the effectiveness of these two methods in the context of present socio-economic scenario of India and
- 4) Arrive at a conclusion that how far this non-conventional method i.e. Psorinum Therapy has the scope for advancement irrespective of socio-economic condition.

The predictions of advanced medical scientists all over the world proclaim the impending growth of cancer as an epidemic within the next decade i.e. by the year 2020. Statistical data reveals that people residing in Asian countries comprising mainly China, India, Pakistan and Bangladesh will be the main victims of this catastrophe. Unless a uniform, acceptable, scientific methodology of treatment of cancer which is at the same time economically affordable to a greater mass emerges out within this period, the entire human civilization will be in jeopardy. This calls for implementation of some pre-emptive measures to avoid the inevitable disastrous consequences.

Incidentally the fascinating slogan of cricket loving people of today echoes the voice “Enjoy the 20-20 matches with pomp and grandeur but make sure to build up a solid defence to guard against the possible ignominious defeat. Similarly to initiate the fight against the impending epidemic of cancer the whole world should understand that unless a cost effective and labour intensive method based on economic affordability to fight against CANCER is introduced, the poorer section of cancer patients are destined to ruin in near future i.e. within the year 2020.

The model of cancer research and treatment centre with Psorinum Therapy as suggested in this publication with necessary amendments may offer a tentative solution on this score. The major obstacle to launch a fight against Cancer in India and abroad is not only the absence of a proper method and medicine which can cure the disease but the bare truth lies in the fact that the entire research work as regards diagnosis and treatment of modern Cancer management have been initiated and performed on the assumption of availability of highly expensive technical and sophisticated resources prevalent in developed countries. Even then the conventional method till date suffers from serious limitations and cannot offer a complete and fully curable treatment for multiple varieties of Cancer. The patients whose health performance status is below fifty(50) as per Karnofsky scale, those who have lung effusion, who are resistant to Radiotherapy and Chemotherapy, who cannot withstand toxic drugs and terminal patients unable to take resort to this cost prohibitive and highly technical

methods of treatment are automatically eliminated from its domain. In fact it provides certain partial measures like Surgery, Radiotherapy and Chemotherapy either solely or in conjunction with one another and invariably with limited success. The inability to eradicate the disease entirely by Surgery, Radiotherapy and Chemotherapy and being restricted only to patients having physical condition within tolerable limits has in fact prompted to make a thorough search for unconventional drugs.

Without raising any controversy, it must be accepted that with the advancement of science, the improvement in conventional therapy by judicious combination of Surgery, Radiotherapy and Chemotherapy according to the merit of the cases has produced commendable results in certain cases despite some limitations inherent in the system. It would be a prudent step to appreciate that under the present socio-economic scenario in India even if only 5% to 10% of the patients can afford to come under the purview of this highly expensive system, they should get an opportunity for this kind of higher amenities. It would be a great benefit and blessings for those lucky few. But a panoramic view of the entire situation arising out of this dreadful disease proves beyond doubt that no specific drug could be found as yet in spite of prolonged big budget research works throughout the world.

In developed countries, however, the Cancer management system has to some extent been stream-lined and early detection of the disease of Cancer has become a reality with the help of a large number of specially trained General Practitioners who after examining the patients, send them directly to onco-surgeons whenever doubt arises in their mind. Onco-surgeons in their turn collect the related tissue and send them to Pathologists and the patients undergo all Radiological tests which help for early detection of the disease. Practically onco-surgeons lose no time in surgical cases. By this effective process of screening, only the advanced inoperable cases are left out and others are referred to Oncologists for necessary radiotherapy or Chemotherapy.

The situation in India is quite opposite, different and just confusing. Besides the high cost involved in the Conventional treatment the total Cancer management in India is being tackled mainly by an Oncologist. The Onco-surgeons are so inadequate in number that in many cases General Surgeons having insufficient Knowledge about the disease merrily proceed with surgery. These General Surgeons are neither so experienced nor having such precision like the Onco-surgeons who are well conversant of keeping stipulated margins while debulking malignant growth and damaged cells or plucking out lymph nodes in different parts or organs of the body. As a result in a number of cases avoidable post-operative complications arise and Oncologists have to face the precarious conditions of the patients single-handedly.

To tell the truth, what an unbelievable predicament an Oncologist is confronting today in the socio-economic scenario of an under developed country like India. In case everything goes fine which postulates that successful debulking of the malignant growth was done through surgery and maximum 30 sessions of Radiotherapy has been conducted smoothly for more than a month and thereafter all the six cycles of Chemotherapy could be administered successfully even then the entire exercise consumes a period of six months only. If the patient's

survival span becomes 5 years on average then the job of the Oncologist virtually comes to an end within six months and the patient has to depend solely on the mercy of the General Physicians for the remaining days of his life. The number of general physicians in the under developed countries is of course very rare who are well conversant with the requirement of these types of patients who need timely supportive interventions and palliative treatment for prolonging their life.

Especially in an under developed economy the exorbitant price of the conventional Chemotherapy drugs are so cost prohibitive that majority of the patients fail to afford these drugs. Even if there are few opulent patients who can afford its expenses, many of them are eliminated either on the ground of having physical resistance to those drugs or their health performance status being below 50 as per Karnofsky scale. Under this compelling circumstances in India attention is to be concentrated on 90% of the Cancer patients who are automatically debarred on financial ground from entering in the process of conventional treatment. Unless the entire disease of Cancer is viewed in its totality keeping in mind about the socio-economic scenario of the country, the proper solution cannot be spelt out. It is essentially imperative to strike a balance between the financial condition of the patient and the cost involved in running the treatment for the minimum period of time and more so when the treatment may prolong due to the complexity of the particular type of disease. If adequate funds are not available how the intake of modern costly medicines along with sophisticated methods of treatment together with administration of palliative managements involving blood transfusion, pleural paracentesis, stenting etc. and series of pathological examinations can be conducted? The exorbitant price of the conventional Chemotherapy drugs coupled with a possibility of its induced adverse reaction on patients during the course of treatment practically offers no solution to the vast majority of the people as it goes out of their reach. Especially in the under developed countries it remains well nigh impossible where most of the people live below the poverty line and in the midst of appalling poverty. So, medical counseling becomes evident and an integral part in the treatment of this disease. When certain symptoms resembling Cancer becomes perceptible in the body of a person, he becomes totally disheartened and confused. He does not know what to do, where to go or who will give him the proper advice.

FRANTICAL SEARCH FOR ALTERNATIVE METHODS

In fact, once cancer is detected, the patients in general, consider the treatment of cancer as a mere stop-gap arrangement before the final exit of human soul. In spite of the application of these highly sophisticated improved technologies it could make only a transient halt to the irresistible onslaught of the disease. More over the cost involved for this stop-gap arrangement are also so exorbitant that majority of the victims especially in underdeveloped countries, can rarely afford to come under its purview. As a consequence a large number of patients in desperation frantically searches for alternative modes of treatment in different forms, which are as it had been since the primitive days. These modes had little relation with the pathological findings and were usually applied on trial and error basis. In some way or other most of this forms rely on the inherent healing power of human nature and usually

belong to Homeopathy, Ayurveda or other streams of medical treatment.

ADVANCEMENT OF MEDICAL SCIENCE SHOULD NEVER BE DENIED

The advancement of medical science may justifiably demand that consolidation of gains already achieved in the sphere of Pathology, Radiology and Oncology must be taken care of and the diagnosis of the diseases should be based on Pathological reports and Radiological findings and the aspects of curability of the disease should be also adjudged and evaluated from these reports available during the course of treatment. It is needless to add that where the diagnosis of the disease of Cancer can only be made correctly by Pathological reports, how its remission or recession can be ascertained without getting through these reports. This great incompatibility remains as a stumbling block in accepting the so-called non-conventional treatment as a rational one.

DECISIVE ROLE OF CONVENTIONAL PALLIATIVE TREATMENTS

This study, though belongs to non-conventional method of treatment, has however accepted that basic principle of assessing the disease through diagnostic reports from the very out-set and has made proper documentation of all pathological and radiological reports. This effort has initiated a joint collaboration between two completely divergent groups in the domain of cancer research. The palliative measures adopted by the conventionalist in the treatment of cancer were combined and applied selectively while conducting the treatment with a non-conventional medicine. This complementary and supplementary use of two different streams has almost produced synergic effect to give outstanding results.

Non-Conventional treatment of Cancer gradually gets the right perspective through Psorinum Therapy

This research work was conducted in the state of West Bengal in India amongst more than 500 patients since the year 1980. The source of drug Psorinum, a homeopathic medicine is the alcoholic extract of the scabies, scurf, slough and pus cells and it was administered orally. Prior to this therapy all the diagnosed patients were evaluated and staged by a routine special diagnostic technique. In the formative years when the trial just started, the drug Psorinum was administered without any supportive care. Though considerable shrinkage was observed in case of solid tumour, the mortality remain very high. In the latter stage when the supportive treatments viz. blood transfusion, plural aracentesis, analgesic, bronchodilators, stenting and palliative management were applied in conjunction with the medicine, the mortality rate declined considerably and in many cases better results were obtained.

Re-orientation inside the Human Body Emphasised

Perhaps it will not be out of place to mention that the mode of operation of Conventional treatment decisively aims at debulking the malignant growth by way of Surgery or destruction of uncontrolled cells by way of Radiotherapy or Chemotherapy. On the contrary, the emphasis of non-conventional treatment principally lie on reconstruction or rectification from within. Analytically the Conventionalists deal with Cancer in a way just like other diseases which results from foreign invasion or intrusion of some external agency like Virus, Germs or Bacteria and naturally urges for a stern, decisive and drastic measure like military solution.

On the other hand the non-conventionalist mostly rely on the inherent healing power of human nature and prefers to introduce some catalytic agents which can effectively help to fight out the internal disorder and usher in some steps which will generate a harmonious condition inside the body. This endeavour will set everything in order or will accelerate the repair work or healing up activities of the internal deficiency in a congenial atmosphere within the body aiming at restoration of peace and harmony instead of launching a devastating battle of annihilation against the enemy.

Quality of Patient's Life Improved

In view of the entire scenario which has been stated above this research work has realized the great importance of adhering to the non-conventional medicine Psorinum, the results of which appear to be quite encouraging when 80% patients were found responding to this therapy. Most of the cases showed marked remission of presenting symptoms like pain and respiratory distress. Pain relief was discernible even when bones and nerves were involved. Life was prolonged. Quality of life improved. Haematological picture indicated satisfactory improvement. Significant progress was found in ECOG score. Growth and metastatic lesions completely disappeared in more than 45 cases. Where growth completely disappeared the patients had good disease free survival records. Till date remarkable results have been achieved by this cost effective medicine without any adverse effects on patients on its application and empirical studies has revealed that definitely an anti-cancer ingredient is latent in this drug. But none should lose sight of the fact that Pharmacodynamics and Pharmacokinetics of this drug is yet to be ascertained and it poses a great challenge to the Scientists of the coming days to find out its molecular structure.

Meticulous Documentation of Pathological Reports

During the course of research work all the documentation work was maintained properly. In order to examine the veracity of our findings these were recorded truthfully, examined accurately and scrutinized meticulously. Pathological diagnosis of Cancer from a tissue specimen was obtained prior to administration of alternative medicine in each case. All pathological reports for the diagnosis of the disease as well as radiological reports indicating tumour regression as the case progress was kept according to disease type and location of the site of the disease in the body. The help of eminent Pathologists, Oncologists and Radiologists were secured to have vivid documentation of the Pretreatment and Post-treatment findings objectively with the help of specialists. The histopathology reports, FNAC reports and other reports pertaining to Ultrasonography, CT Scan, X-ray, Haematology etc and all other evidences were maintained from the very beginning.

A Panoramic View Helped to Develop a Strategy of Harmony

Nevertheless, the introduction of these palliative treatments obviously required active participation from the Oncologists, Surgeons and Pathologists. This task of ensuring participation from these personalities from conventional allopathic stream to this non-conventional treatment may at a glance appear to be an absurd proposition. Since the area of activity and the task to perform by both the conventionalists and non-conventionalists are

in many cases common as it is related to regression of malignant growth, it was not easy to inculcate the spirit of co-operation between these contending rivals. By dint of the careful selection of area of operation and by chalking out acceptable strategy and tactics, a situation could be successfully arrived at where the activities of two diametrically opposite groups became complementary to one another. In fact, with the help of a panoramic view only those cases were selected for treatment under this non-conventional therapy which was deemed to be unfit for conventional treatment due to different reasons. These cases were usually beyond the purview of conventional therapy either being resistant to Radiotherapy and or Chemotherapy or becoming below 50 Karnofsky scale in respect of health performance status or the affected sites were considered to be in-appropriate for surgery due to their deep roots or metastasis in the lung or GI tract including Liver, Gall bladder, Pancreas, Stomach etc. Thus, the possibility of a conflict because of in-built rivalry of the two streams of treatment could be carefully avoided. Gradually a coordinated group of benevolent Pathologists, Radiologists, and Oncologists emerged out who were conducting research work in unison along with other experienced Surgeons and Physicians in order to secure all types of palliative treatments necessary to prolong the life of the patients who were under the treatment of this non-conventional medicine.

Objective Documentation Ensured

Incidentally, this study by and large made a probe into those cases where little scope for recovery was left under conventional therapy. But even from this difficult stage very good results were obtained by this alternative therapy. The conditions of the patients were assessed objectively all through and their reports of performance along with statistical data were published in different journals from time to time. Details have also been recorded in the proceedings of National and International scientific conferences.

Changing Scenario in the Medical World for Alternative Treatment

Appreciation or recognition of these studies could not be dreamt of just 25 years ago when the non-conventional drugs as well as the symptomatic treatments were generally looked down upon by the scientific world since it was considered to be hopelessly bereft of science. But things today have radically changed and all the established and leading Cancer Research Institutes are cordially inviting the reports of the positive results, if any, or the findings obtained by the various non-conventional treatments as well. Probably the outstanding results of some of this alternative treatment have softened their earlier rigid stance and have helped a lot to develop a deeper insight in the arena of modern medical science.

Guidance of Knowledgeable Physicians is Essential

Admittedly due to multiplicity of its character and wide range of variations of this disease, treatment of Cancer whether Conventionally or in a Non-conventional way has to be made by a Physician or a Specialist who has attained fair knowledge regarding the diagnosis of the disease, identification of its type and probable survival period of the patient keeping

close watch on his health performance status and the affected sites of the patient.

Constant Assistance of Expert Oncologists and Radiologists must be secured

While an Oncologist or a Radiologist with his fair amount of Knowledge and experience is capable of ascertaining the above features, the physicians who deals with alternative therapy has to essentially acquire this knowledge coming in contact with the great exposure as regards contribution of modern medical science including pathological and radiological findings. Only after embarking upon this strenuous path of clinical experience and assiduous analysis of case studies this arduous goal can be achieved. In this research work 25 years experience with alternative medicine Psorinum Therapy conducted with the help of objective assessment as stated above with a coordinated group of experts in different branches of medical science we could arrive at a stage where it may be reasonably said that Non-conventional medicine Psorinum has emerged out as a scientific therapy of Cancer.

Process of Integration between two different kinds of Treatment will be a great milestone

The facts stated above obviously leads to be conclusion that by inculcating a spirit of cooperation and by fostering an idea of gradual integration between two apparently contending groups viz. Conventionalist and Non—Conventionalist, a great milestone can be achieved to reach the objective. Primarily patients are to be selectively ramified considering his financial standing, health performance status, physical resistance to toxic drugs and other features as well. If it is found that neither his finance permits, nor his health condition allows continuing Conventional treatment, emphasis should be given to Palliative management from the very inception. Arrangement of blood transfusion, drainage of Plural effusion and Ascitis, administering medicine for stoppage of bleeding and relief from pain, feeding through rice-tube, use of Catheter etc. are less expensive methods that can be effectively introduced for prolongation of life even in case of terminal Cancer patients. Simultaneously treatment can be conducted with Non-conventional, non-toxic drugs like Psorinum by which quality of life will also improve. It transpires that by choosing correct strategy and tactics regarding categorization and selection of patients depending on their financial and health conditions, the conflict between the two completely divergent groups of physician on Cancer can be carefully avoided. Ultimately with the passage of time the Conventionalists finding no other alternative for complete cure, specially, in case of terminal patients will be rather compelled to take resort to such process of integration of both the procedures where Non-conventional drug like Psorinum will find its suitable place.

How Psorinum Therapy becomes an automatic choice

In the above context none should lose sight of the fact that the Medical World is not free from the greedy grip of commercial tycoons. The medicines on Cancer used by Conventionalists especially while introducing Chemotherapy courses, leave a gigantic profit margin and the powerful Media of today willy-nilly bestows accolades and encomiums in their favour conniving with pharmaceutical laboratories. On the other hand, low cost Non-conventional drugs are economically less alluring for all these concerns. As a result, sponsors

lack initiative to invest funds in the research works with Nonconventional drugs and consequently the proper research infrastructure in this stream is very difficult to emerge. Irony of fate lies in the fact that Chemotherapy doses, the high valued research products of Western world and the disease of Cancer have become almost synonymous though the treatment with that is out of the reach of most of the people in our land. To save from the eventuality of losing both the life of the patient and the future of his family, relatives run helter skelter as per their financial ability. Guided by laymen commonsense they apply indigenous methods, approach quacks and rushes for unscientific, superstitious drugs that their pocket permits. In order to be rescued from this miserable plight of a large number of people, research for less-costly drugs become absolutely imperative. The only silver lining in favour of this Non-conventional research work is the poignant necessity to invent such remedy which will be less costly and affordable by a large section of the people say 90% of the patients in our country who are abjectly pauper and are unable to undergo treatment with Conventional drugs due to more than one reason stated earlier. However due to availability of abundant terminal patients and large sample size, the clinical trial with Psorinum has helped tremendously to open a new horizon. These trials which are clinically examined and attended by a group of renowned Physicians and Surgeons under our guidance in the institute of Critical Cancer Management Research Centre and Clinic at Kolkata with written informed consent of the patients has its foot firmly planted on their own ground. It is always correctly reiterated that unless research work is on a realistic basis by the son of soil with his available resources in the midst of the crisis, the problems will never get its solution.

Determination, Perseverance, Courage is necessary to remove in-built resistance against research work in this land of superstition and ignorance

It is well known that diagnosis and treatment of Cancer on a scientific basis has always been initiated by the economically advanced countries of the Western world. Therefore, the method and infrastructure of research in Cancer in our country has always been a matter of neglect and remains far behind from the desired level. The persons in our country who could be visionaries to carry forward these tasks were unfortunately lacking in confidence and initiative and perhaps do not believe that research works in Cancer is at all possible with so meager resources in our country. Practically, there exists no Infrastructure to evaluate the efficacy of these research works which are being carried out honestly by a few pioneers in our country. It means that if any positive outcome of these research works is available, there is no modus operandi to assess the same. Only these findings will have to be processed through the channel prescribed by foreign authorities and approval from international seminars, conferences and journals may be only a stepping stone in this regard. So the stumbling block in carrying out research program in our country have to be removed by the grim determination and indomitable courage by our research workers themselves who are engaged in this activity totally with their own initiative and enthusiasm. To conduct research works especially with nonconventional methods to fight against Cancer, there are some inbuilt resistances among people at large including scientific workers due to the Academic rigors of scientific research. Nobody dares to shun the age-old tradition of religiously following Western Guidelines

and to pursue new and original uncharted path, which may be a more suitable method to adopt in an underdeveloped country like India. Short-term laboratory based studies may be incorporated to pilot the outcome monitoring eco-friendly mass clinical trial. This innovative enterprise should be made very realistically and should be based on objective report of the health centers which will be intertwined with the progress of day to day development of this therapy. An element of translational research should be instilled in this process so that it can literally move from scientist's bench to patient's bedside. In this process with an unbiased mind endeavour will always be made in fostering collaboration with other countries and advancing the science for the betterment of humanity. It will not be out of place to mention that in a number of cases, remarkable results were shown and efficacy of Psorinum therapy in reducing the intensity of this disease has been proved beyond doubts in many national and international conferences.

A Small Note : Back in the 1980s, in the backdrop of the prevailing socio-economic scenario, I had tried to contribute in whichever way possible towards the treatment and cure of cancer. Today after 36 years I have been able to give a proper shape to my thoughts. To attain this knowledge I had to put in untiring efforts in these years. I had to sacrifice the quality time with my family and near and dear ones. Again I realised that this is not only my problem, but my family had also taken up the challenge and unending support poured in from their end. With time small Aradeep also took deep interest in curbing out the disease from its root. This is not possible for one person to look for a resolution to this massive task. I had to understand the basic idea behind this and gradually a model has grown up. This module is not limited to the patient, the drug or the doctor, but its scope lies beyond all this. The problem is the full-proof treatment plan to cure cancer is not out yet. The treatment that is available is not affordable to many and hence does not attract masses. Again it is not possible for someone to dug deep into the subject and extract everything out of it. But in my case I had to learn everything from the very scratch. I know that this might seem repetitive but for centuries, though several developments have come up in the sphere of cancer treatment, but the ultimate treatment plan and its proper management is yet to be discovered. This begins from the detection of the disease and ends with the death of either the disease or the sufferer. If someone feels interested in doing something new and innovative then firstly he has to learn the management part. There are several steps that needs to be taken care of. To overcome these steps doctors, scientists all over the world are trying day-in and dayout. This effort often turns out to be fruitful as well. Every moment a new drug is getting invented, a new surgical technique is coming up. Cancer does not only mean death. It also implies overcoming certain obstruction along with pain management. If someone gets engaged in the cancer treatment without the proper knowledge of these subjects it would be very hilarious.

I am presenting here two of my papers which would focus more on the management aspect of the disease. I have tried to explain the problem and also to come up with a resolution. Our aim has been to rebuild the entire treatment structure and add out treatment model with it. Keeping this in mind we collaborated with some renowned oncologists to come up with these two papers. The writing that followed by these papers would reflect what idea we had attained in due course. I have tried to put every event in a chronological order so that the reader can relate with the disease, the symptoms, its development and the treatment method to be perused.

Earlier I did not have the idea that any scientific paper or thesis paper is accompanied by an article review. I always tried to present my papers in such a way that it would be understandable for those people as well who do not come from proper medical background. I have tried to make my work acceptable to others and in some way or the other would serve the entire mankind in future. I will behappy that today my work is getting proper recognition and acceptance from all scientific forums and I am thankful for this, especially to Dr. A. K. Varma under whose initiative many of my works received proper exposure.

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NORTHERN BOOK CENTRE
NEW DELHI

THE MANAGEMENT OF CANCER IN TOTALITY - INDIA CAN TAKE A LEAD (In Theory and Application)

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Introduction

Cancer is growing in an epidemic manner all over the world. It demands a highly sophisticated diagnostic approach, treatment and post treatment management with a high level of intersectorial coordination. With the advancement of science there are vast improvements in conventional cancer therapy i.e. Surgery, Radiotherapy, Chemotherapy and the judicious combination of them according to the merit of the cases. But, there are still some limitations. Firstly, these modern sophisticated management techniques are highly expensive and demand expert knowledge for their functioning, which is a great obstacle for cancer management in a developing country like, India. Secondly, some types of cancer become resistant to the above mentioned conventional measures for different reasons; thirdly, in some cases RT and CT can't be continued for the poor physical status of patients. And lastly, but not the least, these modern management centers are urban based, which in turn causes a great problem for management in India. In this perspective, we have taken the opportunity to fill up these gaps with 'Psorinum' therapy as the backbone with the assistance of conventional palliative managements. Among surgical palliative management, like tracheotomy, duodenal jejunostomy, stenting, pleural fluid tap, peritoneal fluid tap etc. and among medical palliative management, infection control, maintain electrolyte balance and nutrition etc. are very important. These set up can be established in the peripheral regions also with slight improvement of existing infrastructure. And as the backbone of treatment approach, we can add 'Psorinum' with the palliative managements for the additive action. Vast population in India is below poverty line and rural people can take this opportunity easily to prolong their life, both qualitatively and quantitatively. Basically, homeopathy treatment is symptomatic. But in our study 'Psorinum' is used particularly for the treatment of cancer based on pathology. This scientific approach, made the study acceptable

of both the users and scientific forum. Moreover 'Psorinum' therapy is less expensive and least toxic. Taking all these things into consideration, if our general cancer management approach emphasizes on 'Psorinum' therapy with the assistance of modern palliative management techniques in cancer management sector, India can take a big step forward.

Objectives:

- To study the pattern of treatment approached with 'Psorinum' therapy with the assistance of modern palliative management techniques.
- To study the quality of life of cancer patients having this medication.
- To study the affordability and acceptability of this therapy to different sections of people.
- To study the 'MODEL' rehabilitation centre for the affected families, with minimum resources.

Material and methods

DRUG : The drug 'Psorinum' is the alcoholic extracts of the scabies scrub, slough and pus cells. 1) The drug administered orally. In addition, another two drugs cheledonium and carduas are also administered orally particularly in cases of liver cancer. These two medicines have the potentiality to improve liver function test.

Larger sample size : Previously we conducted more than one randomized trials takings = 300 patients. (1) This time, we considered 500 patients of different types of cancer having different economical background from different states. Patients are mostly of stage III and IV; also some patients are of stage I and II.

Investigations : Before administering the drug, the cases are diagnosed by different investigative methods, like, skiagram, Ultra sonogram, Histopathology etc. Some patients had taken conventional RT/CT previously. We have also considered the other biomarkers like billirubin haemoglobin etc.

Management : As we stressed earlier in the introduction section, during the administration of the drug 'Psorinum', we have taken the assistance of conventional palliative management technique, as and when required.

Methods of data collection:

- Observing the patients clinically.
- Interviewing the patients directly.
- Record review. Comparing the previous reports with the follow up reports after giving the medicine for a considerable period of time.

Results:

The outcome of the study is encouraging. The effectiveness of 'Psorinum' therapy is very good as we saw in our previous different studies (i), (ii). The patients of stage I and II (i.e. above Karnofsky scale 50) are also getting benefit from this medication. Most of the patients are being systemless (pain respiratory distress), the percentage of reduced tumour size and the improvement of bio – makers (Billi, HB %) is also encouraging very much.

Discussion

We launched our first step to fill up the gaps of conventional treatment of cancer approach. Over the period of time, we observe the betterment of quality of different cancer patients in different randomized trials. Mainly the complementary and alternative medicines (CAM) are symptoms based treatment, but difference between CAM and 'Psorinum' is, in its scientific approach. 'Psorinum' Therapy is mainly pathology based. In our preliminary period, we used to take only those patients, whose predicted prognosis were very poor (below and patients Karnofsky Scale 50) and those patients who were resistant to RT and CT. Those were guided by renowned oncologist of Kolkata. we did not take the cases of renal cell carcinoma and melanoma from the very beginning. did not take cases of CA breast We also, CA ovary, CA Vocal chord etc, where the conventional treatment i.e. surgery RT/CT and combination of them are good enough. Therefore, we never enter into the area of conflict in any sense. Rather, we took the so-called lost cases recognised by our expert oncologists. In this process, we got the opportunity to find some financially handicapped cancer patients of different types, who were above 50 Karnofsky Scale (i.e .stage I and stage II). They also got very much ful result and this success 'Psorinum' Therapy made their life better both qualitatively and quantitatively. To do these, we always took the valuable guidance of the renowned oncologists and also the help of conventional palliative management. This 'Psorinum' therapy is less expensive and least toxic, therefore acceptability and affordability of the medicine of the patients is very encouraging. In a developing country, like India, majority of the people are economically not sound at all and also rural based. Therefore, though the Govt. is trying its level best in cancer detection and management, still, there is a huge scope of this 'Psorinum' treatment approach to deal with the obstacles, we are facing today.

In cancer detection and treatment, the intersection-ordination must be up to the mark. And if we take 'Psorinum' therapy as a nucleus in a generalised manner and the palliative management techniques, as supportive measure, then most of the cancer patients can take the same opportunity with minimum expense with better effectiveness. This approach can change the total scenario of cancer management sector in India. But this should not be one-man army job. Oncologist, Radiologist, specialists in other sector should do the job in co-ordinated fashion all the time.

With the advancement of time, it has become possible to form a set up to manage terminally ill cancer patients in Subodh Mitra Cancer Hospital and Research Centre, Kolkata -700106, by this 'Psorinum' therapy approach, and it has been running with great success. Recently a cancer rehabilitation centre named, 'SEBA NIBAS' Kolkata -700048, has been established for the poor cancer patients from remote places in West Bengal and India and this set up has become possible purely with the help of common people physically, mentally and also financially. They became convinced and came together with a big heart to fulfil the dream. This urge and awareness of common people is essential very much, vast as the real problem is one. In addition, Anti-Tobacco movement has been incorporated with this centre. This total 'MODEL' can be flourished and extended all over India.

CONCLUSION

Some limitations are also there. We do not know much about the Pharmacokinetics and Pharmacodynamics. A big scientific exploration is needed for this. Many more scientific 'acid test' required for the well recognition of 'Psorinum' to general population. People are still unaware about the medicine. This scenario should be changed as early as possible. The intersectorial Co-ordination in this Allopathy system and Homeopathy system is also of great concern. This is not easy task to do. Govt. and NGO's should also pay their kind consideration regarding this. Above all, more and more scientific research works from different angles of view should be conducted to make it popular in general. For sake of the betterment of mankind it is very much urgent. As we have said, we are trying and succeeded to a great extent to involve the entire population within our reach in the matter. As a result, the patients coming from the poorer sections are getting best possible 'Psorinum' treatment without spending a farthing.

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The school of tropical Medicine, Calcutta, performed the leading role in this research work & Dept. of Radiotherapy of Medical College Hospital at Calcutta, extended their whole – hearted cooperation. Invaluable Service was rendered by Dr. Dipankar Das Gupta who had been Associated with the Dept. of critical Management of tata Memorial Hospital at Mumbai. We are grateful to Subodh Mitra Cancer Research Institute for extending their hands of cooperation for the practical application of our 'MODEL' approach. We are thankful to Mrs. Ramala Chakraborty, Mr. Asoke Basu and Mr. Pannalal Guha Thakurta of SUBODH MITRA CANCER RESEARCH INSTITUTE for their whole hearted cooperation.

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TOBACCO COUNTERS HEALTH

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PSORINUM MAKES A MAJOR BREAK THROUGH IN THE TREATMENT OF TOBACCO RELATED LUNG CANCER

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Introduction

Tobacco has serious harmful effects on some vital organs of the human body. Lung is the most affected part of them. In spite of the growing awareness that is being generated throughout the world against the ill effects of Tobacco, its use cannot be abandoned in a day. It will take a long course and eventually occurrence of Tobacco related diseases like lung cancer cannot be ruled out abruptly. This calls for implementation of some pre-emptive measures to avoid the inevitable disastrous consequences. It is needless to mention that besides other factors, smoking Cigar, Cigarette and Bidi is primarily responsible for Lung Cancer. The escalation of this disease among the vast majority of poorer section of people in underdeveloped countries takes place mainly due to their popular habit of smoking Bidi, but unfortunately most of them cannot come under the purview of conventional methods due to inadequacy of their funds and other constraints as well.

Aim of the study

A panoramic view of the entire situation arising out of this dreaded disease proves beyond doubt that no specific drug as yet could be found out despite prolonged research work throughout the world. Till date the conventional method of treatment cannot offer a complete and fully curable treatment for the multiple varieties of cancer. The patients whose health performance status is below 50 as per Karnofsky scale, those who have lung effusion, who are resistant to R.T. and C.T., who can not withstand toxic drugs and terminal patients unable to take resort to this cost prohibitive and highly technical methods of treatment are automatically eliminated from its domain. In fact it provides certain partial measures like Surgery, Radiotherapy and Chemotherapy either solely or in conjunction with one another and invariably with limited success. The inability to eradicate the disease entirely by Surgery or Radiotherapy and the application of Chemotherapy being restricted only to the patients having physical condition within tolerable limits has in fact prompted to make a thorough search for unconventional drugs.

Keeping in conformity with cancer management, Psorinum, a homeopathic medicine

was selectively applied, matched and administered to a number of terminal cancer patients with care and caution, which brought out remarkable results and incidentally without any adverse side effects (Chatterjee A.K. etc. al. 1993, 1995 and 1999).

Material and methods

Drug

The drug Psorinum, a Homeopathic medicine, is the alcoholic extract of the scabie's scrub, slough and pus cells. The drug was administered orally, approx. 0.01 ml/kg body weight per day as a single dose in empty stomach.

Investigation

Prior to the therapy all the diagnosed patients were evaluated and staged by routine and special diagnostic techniques including skiagraphy, barium meal, fiberoptic endoscopy, bronchoscopy, ultrasonography, whole body scan and detailed haemogram. The histopathology was re-examined in all the detected cases of carcinoma and each case irrespective of clinical stage was evaluated by oncologist. In most of the cases the oncologist, considering the grim prognosis, did not recommend conventional therapy.

Patients

The drug is tried in 80 lung cancer cases [12 small cell and 68 non-small cell]. All the cases were histopathologically confirmed, terminally ill with very low general condition of health. The trial was conducted mainly in the state of West Bengal and the patients were mostly Bengalees with few people belonging to other states as well. Patients, suffering from carcinoma of different organs in stage III & IV and performance status (ECOG) of 3 or 4 were taken for study as a randomized open trial after obtaining consent from them.

Management

Along with Psorinum therapy symptomatic therapy for control of infection, haemoptysis, thoracentesis, analgesic, bronchodilators, blood transfusion and nutritional support were given. Radiotherapy and Chemotherapy were not given excepting a very few cases where help of RT was taken for relief of unbearable pain due to erosion of bone.

Assesment

Tumour size shrinkage was regularly assessed by physical examination, skiagraphy, and roentgenogram, Sonography or CT scanning for accurate evaluation of size.

Results

The results are quite encouraging. 80% patient found responding to Psorinum therapy. Most of the cases showed marked remission of presenting symptoms like pain and respiratory distress. Pain relief was discernible even when bone and nerves were involved. Life was prolonged. Quality of life improved. Haematological picture indicated satisfactory improvement. Significant improvement was found in ECOG score. Original growth and metastatic lesions completely disappeared in one small cell and 9 non-small cell carcinoma. Where growth completely disappeared the patients had good disease free survival records.

No adverse side effect of the drug was noted. Relevant table is illustrated as follows.

Types of Cancer	No. of case Studied	Male	Female	Mean Survival Up to 24 months (%)	Tumour Completely Disappeared
small cell	12	9	3	8.3%	1
non-small cell	68	56	12	42.6%	9

Advantages

The drug can be administered orally. In comparison to other drugs for chemotherapy it is much less expensive and non-toxic as well. If it is administered in conjunction with supportive treatments like blood transfusion, abdominal or pleural paracentesis, bronchodialators and stenting of hepato-pancreato-biliary system and relevant surgeries as the case may be, the mortality rate of the cancer patients will decline considerably.

Discussion

In this paper, effectiveness of Psorinum therapy experienced in some of the Tobacco related Lung Cancer cases have been highlighted. But none should loose sight of the fact that when metastasis sets in, the disease may spread over different parts of the body. So during the treatment of lung cancer apart from ensuring the assistance of a Cardio thoracic surgeon, the help of the Gastroenteriologist, the Neurologist and the Orthopedic Surgeons may be required at any moment when the disease spreads into stomach, brain or bone respectively. So the problem should be viewed in its entirety. This therapy was implemented in some varieties of cancer. It could be safely given to patients of older age group where the treatment outcome and prognosis is very poor with conventional therapy. Detailed investigation is required to know how and why the entire tumour is slowly disappearing in some of these cases. Also the pharmacokinetics and pharmcodynamics of this therapy is yet to be ascertained which is already in process. In spite of certain limitations under this unconventional method of treatment our view will be more pronounced if the entire present day situation is brought into proper perspective. To tell the truth, what an unbelievable predicament an oncologist is confronting today in the socio-economic scenario of an under developed country like India. In case every thing goes fine which postulates that successful debulking of the malignant growth was done through surgery and maximum 30 sessions of radiotherapy has been conducted smoothly for more than a month and thereafter all the six cycles of chemotherapy could be administered successfully even then the entire exercise consumes a period of six months only. If the patient's survival span becomes 5 years on average then the job of the oncologist virtually comes to an end within six months and the patient had to depend solely on the mercy of the general physicians for the remaining days of his life. The number of general physicians in under developed countries is of course very rare who are well conversant

with the requirement of these types of patients who need timely supportive interventions as mentioned earlier for prolonging their life.

Especially in an under developed economy the exorbitant price of the conventional chemotherapy drugs are so cost prohibitive that majority of the patients fail to afford these drugs. Even if there are few opulent patients who can afford its expenses, many of them are eliminated either on the ground of having physical resistance to those drugs or their health performance status being below 50 as per Karnofsky scale. Perhaps it will not be an exaggeration to say that this research work has opened a new horizon for general acceptance of this non toxic, economically viable drug especially in under developed countries. Incidentally this research work was done in West Bengal, one of the states of Indian subcontinent, regarded as one of the integral part of the countries having underdeveloped economy in the world with its limited resources of advanced technology, men, money and material.

But success of this project was achieved mostly amongst the patients whose health performance status was below 50 as per Karnofsky scale, which is regarded as the most dangerous stage of cancer. In many cases the trials had to be executed in the dwelling places of these mostly poor and helpless patients below poverty line. An environment full of mistrust and disbelief,, superstition and social antagonism prevailed all round. Eventually to overcome these formidable obstacles, the effective help of 3 NGOs consisting of people form different walks of life had to be secured. The benevolent assistance of distinguished oncologists, Oncosurgeons, radiologists, neurologists and specialists in chest, medicine and other branches of medical science were also made available.

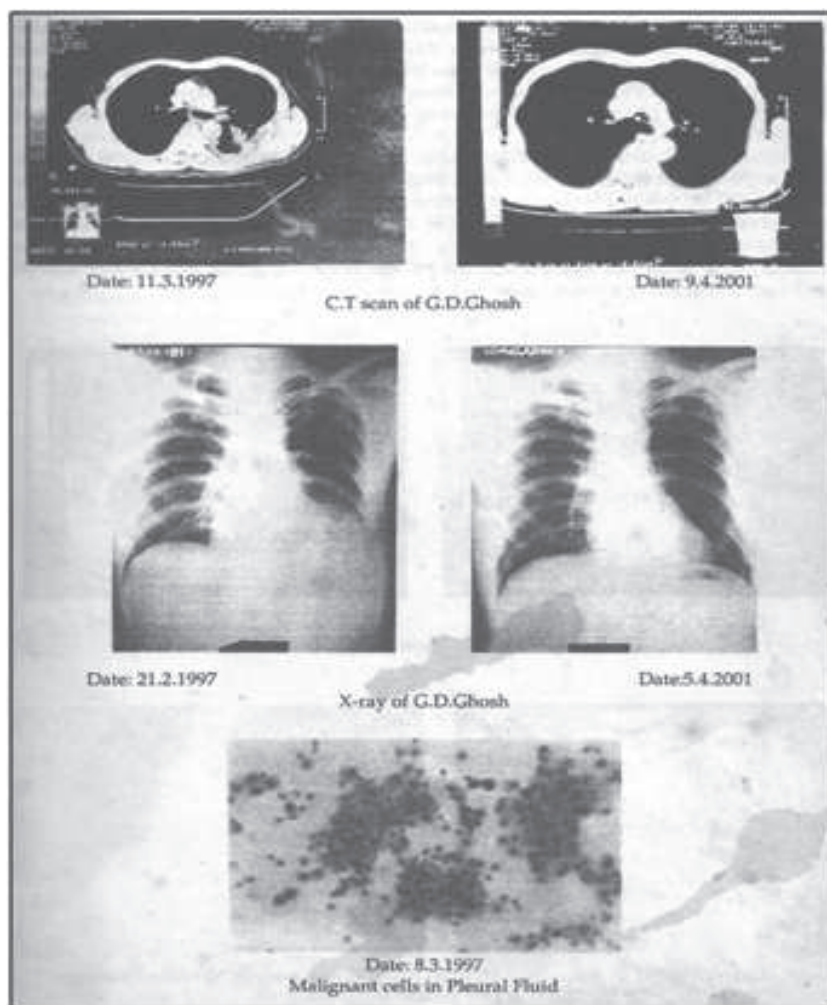
Conclusion

This clinical study affirms that a high power anti cancer agent is latent in the origin of Psorinum found in the scrub, pus & slough from the wounds caused by scabies. To identify that very particular molecule ceaseless research works are on progress with the alcoholic extract of these products but greater scientific efforts may be necessary to make it a resounding success. Nevertheless we must be aware of few limitations associated with this Psorinum therapy. Firstly, to match a Homeopathic medicine in an Allopathic management based on pathological findings is no doubt a very complex proposition. Secondly, unless Doctors belonging to Allopathic stream agree to extend their hands of co-operation to assist in their respective areas, the total management of the disease can not be effective simply by administering the medicine orally. Whenever crisis will arise palliative Allopathic treatment will be necessary. Thirdly, until and unless Psorinum therapy becomes widely known and accepted by the scientific world, the patients may be drifted away to other conventional methods without knowing their inherent limitations. Fourthly, usually no arrangement exists in the Government Hospitals for management of terminal patients and to continue this treatment in a Nursing Home is too much expensive. So in majority of cases, a minimum set up for crisis management for terminal patients had to be arranged in their dwelling places. Lastly & finally, after this research work and greater scientific efforts the medical world should be kind enough to accept this Psorinum therapy as one amongst many successful methods of treatment of cancer.

Case study reports

Case No. 1

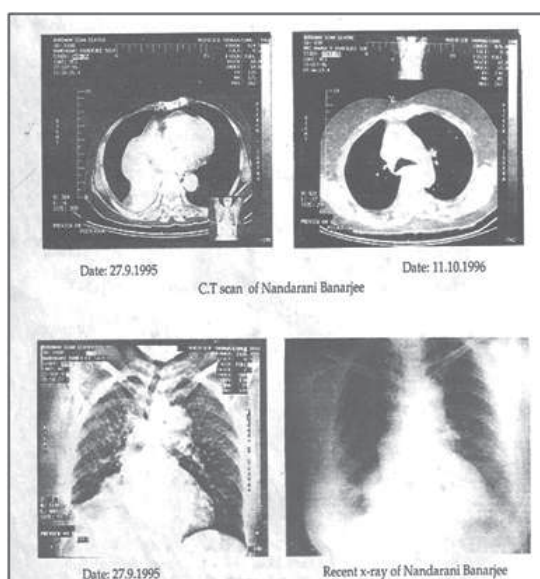
Govinda Das Ghosh, 70 years, Male, Ex-College Teacher, chain smoker, was presented with cough, respiratory distress and severe chest pain on 14.02.1997. X-ray report revealed homogeneous opacity and effusion in left lung. Pleural paracentesis was done in S.S.K.M. Hospital, Cal. And pleural fluid showed presence of fair no of malignant cells (Adeno Carcinoma). C.T. scan on 11.03.1997 detected effusion and SOL in left lower zone. Radiotherapy could not be applied due to effusion and Chemotherapy could not be done for poor general health. Psorinum therapy started on 20.03.1997 with other supportive and symptomatic management. Gradually SOL and effusion completely disappeared with Recent X-ray and C.T. scan shows no abnormality in Lung. The patient completely and after 5 years, actively engaged in a prominent role in the anti-tobacco movement organized by our society.



Slide show with visual representation

Case No. 2

Nanda rani Banerjee, 50 years, female house wife, addicted to tobacco chewing habit in the form of taking Zarda & Tobacco dust in teeth and exposed to passive smoking due to 30 years acquaintance with her chain smoker husband, was presented with chest pain, dispnoea & haemoptysis. X-ray of chest was done on 24.09.1995 showed widening of mediastinum, obliteration of Rt. Para Celiac region. C.T. scan on 27.10.1995 detected Rt. Perihilar intrapulmonary mass with collapse consolidation of middle & Lower Lobe and pretrachial lymphadenopathy. Bronchoscopic biopsy on 18.11.1995 diagnosed small cell carcinoma. A course of Radiotherapy & Palliative Chemotherapy started on 17.02.1996 but could not be completed due to deterioration of general health (Calcutta Medical College Hospital – Ticket No. 951616). Psorinum therapy was started on 09.05.1996. Patient gradually improved which showed marked reduction in size of the mass and total resolution collapse consolidation. 6 years has elapsed and patient is having good, disease free survival record and now leading a normal life.



Slide show

Acknowledgements

The School of Tropical Medicine, Calcutta performed the leading role in this research work & Department of Radiotherapy of Medical College Hospital at Calcutta, extended their whole-hearted cooperation. Invaluable service was rendered by Dr. Dipankar Das Gupta who had been associated with the Department of Critical Management of Tata Memorial Hospital at Mumbai. Dr. Amiya Kumar Hati, exdirector School of Tropical Medicine, Dr. Hiranmoy Mukherjee and Dr. P.K. Kundu of the same institution and Prof. R.N. Bramhacharya, Ex-Prof & Head of the Dept. Radiation Oncology of S.S.K.M. Hospital were constant sources of inspiration. Prof. Dilip Bose, Vice-Chancellor, Tripura University & Ex-Secretary of Directorate of Science and Technology, West Bengal, immensely helped to conduct the research work in different Branches & Departments ignoring so called institutional formalities. We are

thankful to Sri Pushpen Bhowmik, Sri Tapan Sen & Sri Aradeep Chatterjee for their sincere efforts for the compilation of data in this regard.

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* *Proceedings of World Assembly on Tobacco Counters Health – 29th Sep to 3rd Octo 2002, Tobacco Counters Health – Volume 2 , Editor – A.K. Varma, Macmillan*



From left to right Dr. Aradeep Chatterjee, Nandarani Banerjee and Dr. Asim Chatterjee



Gobindo Das Ghosh to the extreme right

TOBACCO COUNTERS HEALTH

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NON-CONVENTIONAL TREATMENT OF TOBACCO RELATED CANCER GRADUALLY GETS RIGHT PERSPECTIVE THROUGH PSORINUM THERAPY

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Introduction

The major reason for the escalation of the dreadful disease like Cancer is inherent in the use of Tobacco in different forms. Despite the world wide movement against the use of Tobacco, its harmful habits cannot be effectively reduced. So the prevention, cure and proper treatment of Cancer is intensely felt day by day. The dedicated groups of research workers engaged in the treatment of cancer all over the world do realize that this dreadful disease is still irresistible and advancing menacingly to become the prime life-taker within decades. Notwithstanding all out endeavour by the entire medical world coupled with ambitious research projects involving billions of dollars throughout the world the out-come is far from being satisfactory. Till date no concrete method evolved out for the protection & cure of the disease. The present stage where the conventional method of treatment has reached is characterized by its obvious limitations. In fact, it is advancing in a limiting manner and now leaving apart the question of curability of the disease, the prime concern is to evolved out a systematic method of scientific treatment and to introduce the same in a uniform way. The modern treatment with its impeccable application of three incontrovertible effective weapons like Surgery, Radiotherapy and Chemotherapy has nevertheless exposed the serious limitations inherent in this treatment. The people at large gets more disillusioned by its inefficacy when they find that a large number of patients within few months of this highly

expensive treatment become resistant to Radiotherapy and Chemotherapy and technically become inoperable and when in some other cases, patients health performance status go below 50 Karnofsky scale.

Frantical Search for Alternative Methods

In fact, once cancer is detected, the patients in general, consider the treatment of cancer as a mere stop-gap arrangement before the final exit of the human soul. In spite of the application of these highly sophisticated improved technologies it could make only a transient halt to the irresistible onslaught of the disease. More over the cost involved for this stop-gap arrangement are also so exorbitant that majority of the victims especially in underdeveloped countries, can rarely afford to come under its purview. As a consequence a large number of patients in desperation frantically searches for alternative modes of treatment in different forms, which are usually symptomatic in nature and largely depends on the clinical inspection of Doctor's eye as it had been since the primitive days. These modes had little relation with the pathological findings and were usually applied on trial and error basis. In some way or other most of this forms rely on the inherent healing power of human nature and usually belong to Homeopathy, Ayurveda or other streams of medical treatment.

Advancement of Medical Science should never be denied

But the advancement of medical science may justifiably demand that consolidation of gains already achieved in the sphere of pathology, Radiology and Oncology must be taken care of and the diagnosis of the diseases should be based on Pathological reports and Radiological findings and the aspects of curability of the disease should be adjudged and evaluated from these reports available during the course of treatment. It is needless to add that where the diagnosis of the disease of Cancer can only be made correctly by Pathological reports, how its remission or recession can be ascertained without getting through these reports. This great incompatibility remains as stumbling block in accepting the so-called non-conventional treatment as a rational one.

Decisive Role of Conventional Palliative Treatments

This study, though belongs to non-conventional method of treatment, has however accepted that basic principle of assessing the disease through diagnostic reports from the very outset and has made proper documentation of all pathological and radiological reports. This effort has initiated a joint collaboration between two completely divergent groups in the domain of cancer research the palliative measures adopted by the conventionalist in the treatment of cancer were combined and applied selectively while conducting the treatment with a non-conventional medicine. This complementary and supplementary use of two different streams has almost produced synergic effect to give outstanding results.

Material & Methods

This research work was conducted in the state of West Bengal in India amongst more than 500 patients since the year 1980. The source of drug Psorinum, a homeopathic medicine is the alcoholic extract of the scabies, scrub, slough and pus cells and it was administered orally. Prior to this therapy all the diagnosed patients were evaluated and staged by a routine

special diagnostic technique. (1) In the formative years when the trial just started, the drug Psorinum was administered without any supportive care. Though considerable shrinkage was observed in case of solid tumour, the mortality remained very high. In the later stage when the supportive treatments viz. blood transfusion, pleural paracentesis, analgesic, bronchodilators, stenting and palliative management were applied in conjunction with the medicine, the mortality rate declined considerably and in many cases better results were obtained.

A Panoramic View Helped to Develop a Strategy of Harmony

Nevertheless the introduction of these palliative treatments obviously required active participation from the Oncologists, Surgeons and Pathologists. This task of ensuring participation from these personalities from conventional allopathic stream to this non-conventional treatment may at a glance appear to be an absurd proposition. Since the area of activity and the task to perform by both the conventionalists and nonconventionalist are in many cases common as it is related to regression of malignant growth, it was not easy to inculcate the spirit of co-operation between these contending rivals. By dint of the careful selection of area of operation and by chalking out acceptable strategy and tactics, a situation could be successfully arrived at where the activities of two diametrically opposite groups became complementary to one another. In fact, with the help of a panoramic view only those cases were selected for treatment under this non-conventional therapy which was deemed to be unfit for conventional treatment due to different reasons. These cases were usually beyond the purview of conventional therapy either being resistant to Radiotherapy and / or Chemotherapy or becoming below 50 of Karnofsky scale in respect of health performance status or the affected sites were considered to be in-appropriate for surgery due to there deep roots or metastasis in the lungs or G.I tract including Liver, Gall bladder, Pancreas, Stomach etc. Thus, the possibility of a conflict because of in-built rivalry of the two streams of treatment could be carefully avoided. Gradually a coordinated group of benevolent Pathologists, Radiologists and Oncologists emerged out who were conducting research work in unison along with other experienced Surgeons and Physicians in order to secure all types of palliative treatments necessary to prolong the life of the patients who were under the treatment of this non-conventional medicine.

Objective Documentation Ensured

Incidentally this study by and large made a probe into those cases where little scope for recovery was left under conventional therapy. But even from this difficult stage very good results were obtained by this alternative therapy. The conditions of the patients were assessed objectively all through and their reports of performance along with statistical data were published in different journals from time to time. Details have also been recorded in the proceedings of National and International scientific conferences.

Changing Scenario in the Medical World for Alternative Treatment

However appreciation or recognition of these studies could not be dreamt of just 25 years ago when the non-conventional drugs as well as the symptomatic treatments were generally looked down upon by the scientific world since it was considered to be helplessly bereft of

science. But things today has radically changed and all the established and leading Cancer Research Institutes are cordially inviting the reports of positive results, if any, or the findings obtained by the various non-conventional treatments as well. Probably the outstanding results of some of this alternative treatment have softened their earlier rigid stance and have helped a lot to develop a deeper insight in the arena of modern medical science.

Re-orientation inside the Human Body Emphasised

Perhaps it will not be out of place to mention that the mode of operation of Conventional treatment decisively aims at debulking the malignant growth by way of Surgery or destruction of uncontrolled cells by way of Radiotherapy or Chemotherapy. On the contrary, the emphasis of the Non-conventional treatment principally lies on reconstruction or rectification from within. Analytically the Conventionalists deal with Cancer in a way just like Virus, Germs or Bacteria and naturally urges for a stern, decisive and drastic measure like military solution. On the other hand the Non-Conventionalist mostly rely on the inherent healing power of human nature and prefers to introduce some catalytic agents which can effectively help to fight out the internal disorder and usher in some steps which will generate a harmonious condition inside the body. (4) This endeavour will set everything in order or will accelerate the repair work or healing up activities of the internal deficiency in a congenial atmosphere within the body aiming at restoration of peace and harmony instead of launching a devastating battle of annihilation against the enemy.

Socio Economic Scenario taken into Account

However, unless the entire disease of cancer is viewed in its totality in the Socio Economic Scenario of the country the proper solution cannot be spelt out. It is essentially imperative to strike a balance between the financial condition of the patient and the cost involved in running the treatment for the minimum period of time and more so when the treatment may prolong due to the complexity of the particular type of the disease. If adequate funds are not available how the intake of modern costly medicines along with sophisticated methods of treatment together with administration of palliative managements involving blood transfusion, pleural paracentesis, stenting etc. and series of pathological examinations can be conducted? The exorbitant price of the conventional chemotherapy drugs coupled with a possibility of its induced adverse reaction on patients during the course of treatment practically offers no solution to the vast majority of the people as it goes out of their reach. Especially in the under-developed countries it remains well nigh impossible where most of the people live below poverty line and in the midst of appalling poverty.

Quality of Patient's Life Improved

In view of the entire scenario which has been stated above this research work have realized the great importance of adhering to the non-conventional medicine Psorinum, the results of which appears to be quite encouraging when 80% patients were found responding to this therapy. Most of the cases showed marked remission of presenting symptoms like pain and respiratory distress. Pain relief was discernable even when bones and nerves were involved. Life was prolonged. Quality of life improved (5). Haematological picture indicated

satisfactory improvement. Significant progress was found in ECOG score. Growth and metastatic lesions completely disappeared in more than 45 cases. Where growth completely disappeared the patients had good disease free survival records. Till date remarkable results have been achieved by this cost effective medicine without any adverse effects on patients on its application and empirical studies has revealed that definitely an anti-cancer ingredient is latent in this drug. But none should loose sight of the fact that Pharmacodynamics and Pharmacokinetics of this drug is yet to be ascertained and it poses a great challenge to the Scientists of the coming days to find out its molecular structure.

Meticulous Documentation of Pathological Reports

During the course of research work all the documentation work were maintained properly. In order to examine the veracity of our findings these were recorded truthfully, examined accurately and scrutinized meticulously. Pathological diagnosis of Cancer from a tissue specimen was obtained prior to administration of alternative medicine in each case. All pathological reports for the diagnosis of the disease as well as radiological reports indicating tumour regression as the case progresses was kept according to disease type and location of the site of the disease in the body. The help of eminent Pathologists, Oncologists and Radiologists were secured to have vivid documentation of the Pre-treatment and Post-treatment findings objectively with the help of specialists. The histopathology reports, FNAC reports and other reports pertaining of Ultrasonography, CT Scan, X-ray, Haematology etc and all other evidences were maintained from the very beginning.

Guidance of Knowledgeable Physicians is Essential

Admittedly due to multiplicity of its character and wide range of variations of this disease treatment of Cancer whether Conventionally or in a Non-conventional way has to be made by a Physician or a Specialist who has acquired fair Knowledge regarding the diagnosis of the disease, identification of its type and probable survival period of the patient keeping close watch on his health performance status and the affected sites of the patient.

Constant Assistance of Expert Oncologists and Radiologists must be secured

While an Oncologist or a Radiologist with his fair amount of knowledge and experience is capable of ascertaining the above features, the physician who deals with alternative therapy has to essentially acquire this knowledge coming in contact with the great exposures as regards contribution of modern medical science including pathological and radiological findings. Only after embarking upon this strenuous path of clinical experience and assiduous analysis of case studies this arduous goal can be achieved. In this research work 25 years experience with alternative medicine Psorinum conducted with the help of objective assessment as stated above with a co-ordinated group of experts in different branches of medical science we could arrive at a stage where it may be reasonably said that Non-conventional medicine Psorinum has today emerged out as a scientific therapy of Cancer.

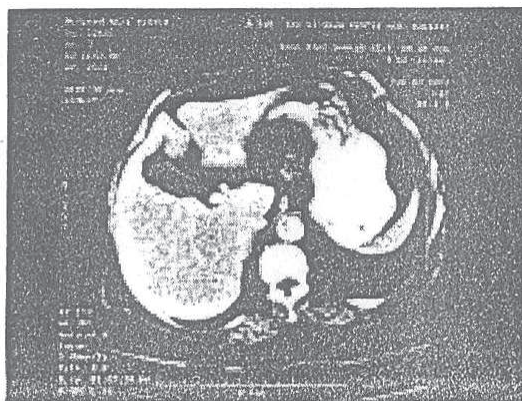
Case study reports

We are forwarding two supportive case studies citing example how the conclusive

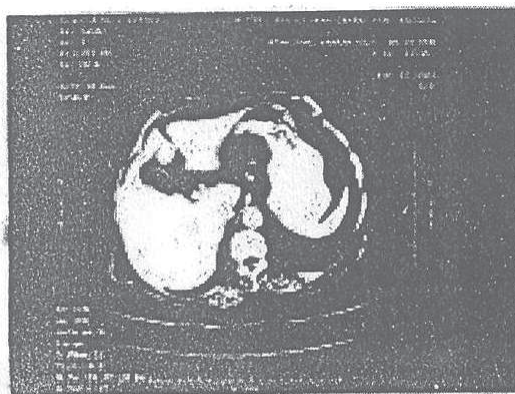
evidence in form of documents were maintained in this research work. The first one being a case of Carcinoma of Gall Bladder with Liver metastasis that underwent a prolonged treatment with Psorinum therapy without Surgery and progressed satisfactorily during last 9 years. While the other is one amongst the recent case studies related to inoperable Ascites metastatic stomach Carcinoma (stage IV) showing marked improvement through this therapy.

Case No. 1

Binapani Sarkar, 51 years, house wife, addicted to Tobacco chewing habit in the form of taking Zarda and Tobacco dust in teeth & exposed to passive smoking for a prolonged period for her acquaintance with her 'chain-smoker' husband, was presented on 25-03-1994 with severe abdominal pain and palpable lump in the right upper zone of abdomen and admitted to Medical College Hospital, Kolkata. C.T. guided F.N.A.C from mass in the right hypochondrium was done on 22-04-1994 which revealed poorly differentiated Adeno- Carcinoma of Gall Bladder. Chemotherapy was suggested but patient developed jaundice subsequently. Chemotherapy was not initiated because of low general condition of health.



Current C.T scan taken on 03.02.2003 showing disappearance of MASS.



Current C.T scan taken on 03.02.2003 showing disappearance of MASS.

Psorinum therapy was started on 05.05.1994 and gradual improvement was observed. The abdomen gradually softened and jaundice disappeared and last C.T scan of abdomen shows no evidence of any primary lesion or any metastasis in liver. Patient is asymptomatic for last 9 years. Chronological C.T Scan & FNAC Photograph of the Patient (C.T Scan of Liver showing presence of Mass. Dated 22.04.1994), (FNAC of the Mass. Dated: 22.04.1994), (Colour microscopic photograph of FNAC slide on 22.04. 94 showing presence of Malignant Cells), Current C.T scan taken on 03.02.2003 showing disappearance of Mass.

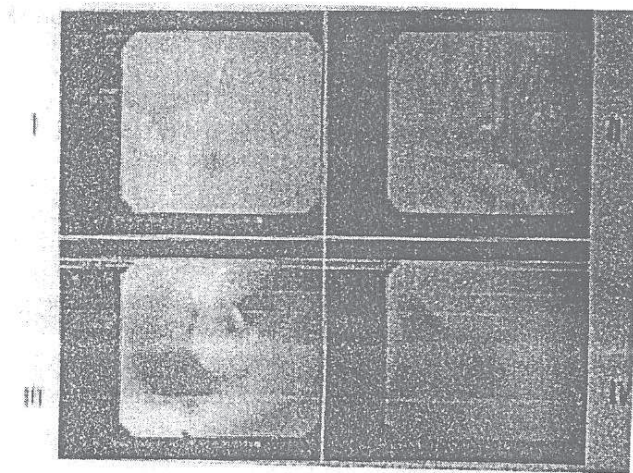
Case No. 2

Mr. Sudhin Mukherjee, 62 years, male a chain smoker and chronic alcoholic was admitted to Sanjoy Gandhi P.G.I Hospital, Lucknow under Dr. Suxene and Dr. G. Roy Chowdhury with severe abdominal pain accompanied with vomiting.

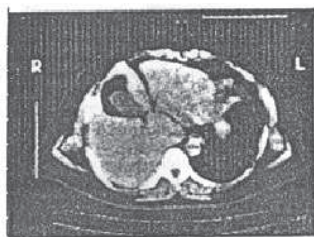
Ref: ticket No – C.R. No 200205074, dept No. 1220, Bed No & Type – 4 pvt, Ward

– B06, Treating Unit – 3, Admission date : 08.03.2002, Discharge Date 30.03.2002, The Clinical Examination revealed (1) a mobile lump in Epigastrium region, (2) Ascites and ulceroproliferative growth in antrum (Adeno carcinoma).

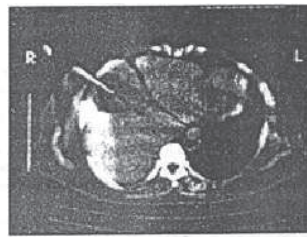
Laparotomy on 14.03..2002



Upper G.I Endoscopy coloured photograph (taken on 26/11/2003)



C.T Scan of Liver showing presence of MASS. Dated: 22.04.1994



F.N.A.C of the MASS. Dated: 22.04.1994



Colour microscopic photograph of F.N.A.C slide on 22.04.94 showing presence of Malignant Cells

Operation Findings

Procedure – Gastro Jejunostomy done but Gastro-enteroectomy was not possible because of large growth in antropyloric region of the stomach infiltrating into D1, hepatoduodenal ligament mesocolon and pancreas. Stomach was dilated and thick walled. Chemotherapy could not be administered due to low ECOG score & adverse Blood report.

On 10.04.2002 the patient was presented through a renowned Surgeon at Kolkata & Psorinum therapy started from 13.4.2002. Patient gradually improved and recent C.T. Scan

reports reveal that Liver, Gall bladder, Pancreas, Spleen and both Kidneys are normal. There is marked thickening of Gastric Antral wall with stranding into the adjoining fat. Stomach bed is clear but no definite fat plane is seen with duodenal cap and adjoining pancreatic head as well as segment II/III of Liver which is otherwise normal. A functioning anterior Gastro Jejunostomy is seen. At present he is clinically fit, takes normal diet, does normal work but unfortunately his previous addiction towards drinking and smoking still persists. Recently, on 28.11.2003, he has been admitted to Onco Surgery department of Chittaranjan National Cancer Institute of Kolkata under the supervision of group of Doctors for clinical study and detail follow up Vide- Ticket. Regd. No. – 5/03/5735. The patient is continuing the Psorinum therapy all through since 13.04.2002 and maintaining a steady progress.

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A Small Note : So far we have been discussing the socio-economic scenario in India and the problem faced by an average man. A panoramic view of the entire situation also leads to microscopic solutions which are on many occasions not feasible in the long run and often is inaccessible. The two papers that I discussed here, both were published in the first decade of this century along with many other papers. But these two require special attention as one of the paper tries to uphold the management aspect of cancer, its treatment and to an extent its prevention, the other one talks about cancer and a drug which has tremendous potential to fight against the disease.

In the early days many people including some renowned doctors as well as scientists misunderstood and protested against me. In the later days when I disclosed what my intentions were they gladly embraced me and made me a part of them. I have written many papers with them in later years as well. Overcoming this obstacle and gaining faith and respect in their eyes has been one of my accomplishments. We have over the years thought over the subject of cancer and have tried to come up with solutions which will not distinguish between rich and poor and will benefit the entire mankind. These two papers uphold the model that I have tried to bring across for so many years and also the promising level of success expected in the coming days.

In the following chapters I have tried to present what cancer is, its several types based upon its origin in the body accompanied by several other conditions. This at times might seem to be highly technical but I have tried to present it in layman's language as much as possible. Following this I have also added a few of the publications, a few of them are under my leadership and a few were carried out under Dr. Aradeep Chatterjee's direction. The later chapters will also show a toddler's journey through many zig-zag ways followed by some case-histories.

CARCINOGEN AND CARCINOGENESIS

Types of Carcinogens:

- Biologic carcinogens (e.g., types of bacteria)
- Chemical carcinogens (e.g., chemicals in tobacco smoke and asbestos).
- Physical carcinogens (e.g., UV radiation)
 - ✓ X-radiation and gamma-radiation
 - ✓ Neutrons
 - Lung cancer
 - Breast cancer
 - Colon cancer
 - Stomach cancer

Process of carcinogenesis:

- Initiation
 - ✓ Mechanisms of oncogene activation:
- Promotion
- Progression

Absorption and metabolism of chemical carcinogens

- Tumor metastasis:
- Tumor angiogenesis:
- Cancer genes:
- Oncogenes:
- Anti-oncogenes or tumor suppressor genes:

Theories of carcinogenesis:

- Gene mutation theory:
- Aneuploidy theory:
- Epigenetic theory

Cellular defense mechanisms in relation to cancer prevention and carcinogenesis:

- Antioxidants:
- DNA repair:
- Role of diet in cancer control:

CANCER AND DIFFERENT TYPES OF CANCER

1. Introduction
2. Biological properties of cancer cells
3. The 6 hallmarks of cancer
 - 3.1. Tumor
 - 3.2. Neoplasm
 - 3.3. Screening
 - 3.4. Diagnosis
 - 3.5. Surgical excision
 - 3.6. Surgical margins
 - 3.7. Grade
 - 3.8. Stage
 - 3.9. Recurrence
 - 3.10. Metastasis
 - 3.11. Transformation
 - 3.12. Chemotherapy
 - 3.13. Radiation therapy
 - 3.14. Adjuvant therapy
 - 3.15. Prognosis
 - 3.16. Carcinoma
 - 3.17. Lymphoma and leukemia
 - 3.18. Germ cell tumor
 - 3.19. Blastic tumor or blastoma
 - 3.20. Adult cancers
 - 3.21. Child cancers
 - 3.22. Infant cancers
 - 3.23. Local symptoms
 - 3.24. Symptoms of metastasis (spreading)
 - 3.25. Systemic symptoms
4. History of cancer
 - 4.1. Cancer in the sixteenth to eighteenth centuries
 - 4.2. Cancer in the nineteenth century
5. Early theories about causes of cancer

- 5.1. Humoral theory
- 5.2. Lymph theory
- 5.3. Blastema theory
- 5.4. Chronic irritation theory
- 5.5. Trauma theory:
- 5.6. Infectious disease theory
- 6. Development of modern knowledge about cancer
 - 6.1. Mutation: chemical carcinogens
 - 6.2. Mutation: ionizing radiation
 - 6.3. Viral and chemical carcinogens
 - 6.4. Hormonal imbalances
 - 6.5. Heredity
 - 6.6. Other causes
 - 6.6. 1. Oncogenes and tumor suppressor genes
- 7. History of cancer epidemiology
- 8. Genetic basis of cancer
- 9. Molecular causes of cancer
 - 9.1. Evading growth suppressors
 - 9.2. Emerging hallmark: evading immune destruction
 - 9.3. Sustaining proliferative signaling
 - 9.4. Enabling replicative immortality
 - 9.5. Enabling characteristic: genome instability and mutation
 - 9.6. Activating invasion and metastasis
 - 9.7. Enabling characteristic: tumor-promoting inflammation
 - 9.8. Inducing angiogenesis
 - 9.9. Resisting cell death
- 10. Process of cancer spreads in the body
 - 10.1. Local spread
 - 10.1.1. Pressure from the growing tumour
 - 10.1.2. Using enzymes
 - 10.1.3. Cancer cells moving through the tissue
 - 10.2. Through the blood circulation
 - 10.3. Through the lymph system
- 11. Why cancers spread where they do

- 11.1. Micrometastases
- 12. Cancer in the twenty-first century
 - 12.1. More targeted therapies
 - 12.2. Immunotherapy
 - 12.3. Nanotechnology
 - 12.4. Robotic surgery
 - 12.5. Expression profiling and proteomics

LEUKEMIA

Discovery of Leukemia

Types of Leukemia

Based on Proliferation

- Acute Leukemia
- Chronic leukemia

Based on kind of blood cell affected

- Lymphocytic leukemia
- Myelogenous Leukemia

Based on the above two groups

- Acute lymphocytic leukemia (ALL)
- Acute myelogenous leukemia (AML)
- Chronic lymphocytic leukemia (CLL)
- Chronic myelogenous leukemia (CML)
- Hairy cell leukemia (HCL)
- Large granular lymphocytic leukemia
- T-cell prolymphocytic leukemia (T-PLL)
- Adult T-cell leukemia

Causes of leukemia

Symptoms

- Blood clotting is poor-

- Feeling weak, tired or generally unwell
- Affected immune system-
- Anemia-
- Bone and Joint Pain-
- Enlarged Lymph Nodes
- Unexplained Fevers
- Abdominal Discomfort
- Headaches and Other Neurological Complaints

Precaution

Treatments of leukemia

- Chemotherapy
- Induction therapy
- Consolidation therapy
- Maintenance therapy
- Stem cell (or bone marrow) transplantation
- Interferon Therapy to Treat Leukemia
- Recovery and follow-up care

Life after treatment

HODGKIN LYMPHOMA

The lymphatic system

Lymph vessels:

- Lymph

Lymph nodes:

- B lymphocytes
- T lymphocytes
- Lymph nodes
- Spleen
- Bone marrow
- Thymus

- Adenoids and tonsils
- Digestive tract

History of Hodgkin's Lymphoma

Causes of Hodgkin lymphoma

Risk factors for Hodgkin disease

- Epstein-Barr virus infection/mononucleosis
- Age
- Gender
- Geography
- Family history
- Socioeconomic status
- HIV infection

Types of Hodgkin disease

Classic Hodgkin disease

- Nodular lymphocyte predominant Hodgkin disease

Classic Hodgkin disease

- Nodular sclerosis Hodgkin's lymphoma
- Mixed cellularity Hodgkin's lymphoma
- Lymphocyte-depleted Hodgkin's lymphoma
- Lymphocyte-rich classical Hodgkin's lymphoma

GENE THERAPY

Contents :

1. Introduction
2. Types of gene therapy
 - 2.1. Germinal gene therapy
 - 2.2. Somatic gene therapy
3. Vectors in gene therapy
 - 3.1. Viruses
 - 3.2. Non-viral methods

4. Procedure of Gene therapy
 - 4.1. Current Clinical Trials
 - 4.2. Future Directions
5. Gene therapy strategies to correct or eliminate cancer cells
6. Cellular immune therapy of cancer
 - 6.1. Genetically engineered tumor cells
 - 6.2. Genetically engineered T lymphocytes
 - 6.3. Protection of hematopoietic stem cells
7. Genetic alteration of cancer cells in situ
 - 7.1. Liposome-mediated gene transfer:
8. Technological hurdles or problems with the gene therapy
9. Deaths
10. Speculative uses for gene therapy
 - 10.1. Gene doping:
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11. Regulations:
 - 11.1. United States:

CARCINOGEN AND CARCINOGENESIS

The term “carcinogen” denotes a chemical substance or a mixture of chemical substances which induce cancer or increase its incidence. Substances which have induced benign and malignant tumours in well performed experimental studies on animals are considered also to be presumed or suspected human carcinogens unless there is strong evidence that the mechanism of tumour formation is not relevant for humans. Classification of a chemical as posing a carcinogenic hazard is based on the inherent properties of the substance and does not provide information on the level of the human cancer risk which the use of the chemical may represent. A carcinogen is any substance, radionuclide, or radiation, also often, but not necessarily, mutagens or teratogens. Carcinogens may cause cancer by altering cellular metabolism or damaging DNA directly in cells, which interferes with normal biological processes. This ultimately results in the formation of a tumour (an abnormal tissue growth) that has the ability to spread (metastasize) from its site of origin and invade and cause dysfunction of other tissues, culminating in organ failure and death. The two primary mechanisms by which carcinogens initiate the formation of such tumours is via alterations in DNA that encourage cell division and that prevent cells from being able to self-destruct when stimulated by normal triggers, such as DNA damage or cellular injury (a process known as apoptosis). There also exist carcinogens that induce cancer through nongenotoxic mechanisms, such as immunosuppression and induction of tissue-specific inflammation. More than 400 chemical agents have been listed as carcinogenic, probably carcinogenic, or possibly carcinogenic by the International Agency for Research on Cancer (IARC), a branch of the World Health Organization that monitors cancer occurrence worldwide and performs epidemiological and laboratory investigations to understand the causes of cancer. Among the carcinogenic substances listed by IARC are a variety of chemical effluents from industry and environmental pollutants from automobiles, residences, and factories. One such example is acrylamide, which is considered a probable carcinogen in humans and is produced as a result of industrial processes and cooking certain foods at high temperatures. It can be released into the environment through its application in wastewater treatment and its use in grout and soil-stabilizer products. Other examples of chemical carcinogens include nitrosamines and polycyclic aromatic hydrocarbons, which are found in tobacco smoke and are associated with the development of lung cancer. Several radioactive substances are considered carcinogens, but their carcinogenic activity is attributed to the radiation, for example gamma rays and alpha particles, which they emit. The different types of potential carcinogens cover a wide range and include both organic and inorganic compounds, radiation exposure from natural or man-made source materials, and living organisms. Any agent that can contribute to cell mutation has the potential to lead to cancer and can be classified as a carcinogen. This often includes many materials that may be harmless in small concentrations, or harmless in the absence of other chemicals that act as triggering agents. Among synthetic chemicals, dioxins have been

called the most toxic chemical compound ever produced by man, and are a byproduct of bleaching in paper mills, from the production of agricultural fertilizers and insecticides, and from incineration. The single smallest, microscopically detectable levels of dioxin have been shown to cause cancer in laboratory animals. Modern industrial processes in the US produce 1,200 pounds (544 kilograms) of dioxin each year, where 12 pounds (5.4 kilograms) alone is enough to count as a lifetime dose for 500 million people. Estimates are that the average American, European, or Canadian already has enough dioxin in his or her body to equal levels that have demonstrated adverse health effects in laboratory animals. Dioxins also act as a cancer enhancer, increasing the intensity of other carcinogens, and are known to contribute to dozens of types of cancer, from skin and liver cancers to Hodgkin's disease.

The most potent natural carcinogen is thought to be Aflatoxin B₁, which is produced by the fungus *Aspergillus flavus* growing on stored grains, nuts and peanut butter, stored in hot, humid environments. Found in rice, soybeans, corn, and wheat as well, it is a potent liver carcinogen causing Hepatocellular carcinoma, which will kill almost every patient that contracts it within one year. It causes cancer by attacking the p53 gene in humans, which works as a tumor-suppressing gene. Certain viruses such as hepatitis B and human papilloma virus have been found to cause cancer in humans. The first one shown to cause cancer in animals is Rous sarcoma virus, discovered in 1910 by Peyton Rous. Other infectious organisms which cause cancer in humans include some bacteria (*Helicobacter pylori*) and helminths (*Opisthorchis viverrini* and *Clonorchis sinensis*).

Radionuclide and radiation sources are also carcinogens. One of the most common, widespread carcinogens in this category is radon gas, which is naturally emitted from trace elements of uranium in soil. Radon gas is the second leading cause of lung cancer in the United States after cigarette smoking, killing an estimated 15,000-22,000 people per year. The World Health Organization estimates that radon gas exposure accounts for 6%-15% of all cases of lung cancer worldwide. Thousands of other potential carcinogens exist in nature and as a direct and indirect result of human industrial processes. Tobacco smoke is known to contain 43 carcinogenic agents, and benzene vapors in gasoline can lead to immune system breakdowns causing leukemia. Dozens of potent carcinogens exist as organic compounds in the average turkey dinner Americans eat for the Thanksgiving holiday. Carcinogens are also found in many cosmetics, as well as in synthetic food preservatives, additives, and coloring agents in the food supply. Cooking protein-rich food at high temperatures, such as broiling or barbecuing meats, can lead to the formation of many potent carcinogens that are comparable to those found in cigarette smoke (i.e., benzo[a]pyrene). Pre-cooking meats in a microwave oven for 2-3 minutes before broiling can help minimize the formation of these carcinogens.

It is essentially impossible to avoid contact with all carcinogenic agents, but, with thoughtful effort and planning, exposure can be greatly minimized.

Co-carcinogens are chemicals that do not necessarily cause cancer on their own, but promote the activity of other carcinogens in causing cancer. After the carcinogen enters the

body, the body makes an attempt to eliminate it through a process called biotransformation. The purpose of these reactions is to make the carcinogen more water-soluble so that it can be removed from the body. But these reactions can also convert a less toxic carcinogen into a more toxic carcinogen. DNA is nucleophilic, therefore soluble carbon electrophiles are carcinogenic, because DNA attacks them. For example, some alkenes are toxicated by human enzymes to produce an electrophilic epoxide. DNA attacks the epoxide, and is bound permanently to it. This is the mechanism behind the carcinogenicity of benzo[a]pyrene in tobacco smoke, other aromatics, aflatoxin and mustard gas.

Cancer is caused by changes in a cell's DNA – its genetic “blueprint.” Some of these changes may be inherited from our parents, while others may be caused by outside exposures, which are often referred to as environmental factors. Environmental factors can include a wide range of exposures, such as:

- Lifestyle factors (nutrition, tobacco use, physical activity, etc.)
- Naturally occurring exposures (ultraviolet light, radon gas, infectious agents, etc.)
- Medical treatments (chemotherapy, radiation, immune system-suppressing drugs, etc.)
- Workplace exposures
- Household exposures
- Pollution

Substances and exposures that can lead to cancer are called carcinogens. Some carcinogens do not affect DNA directly, but lead to cancer in other ways. For example, they may cause cells to divide at a faster than normal rate, which could increase the chances that DNA changes will occur. Carcinogens do not cause cancer in every case, all the time. Substances labeled as carcinogens may have different levels of cancer-causing potential. Some may cause cancer only after prolonged, high levels of exposure. And for any particular person, the risk of developing cancer depends on many factors, including how they are exposed to a carcinogen, the length and intensity of the exposure, and the person's genetic makeup. Exposures to these “agents of evil” can cause some wacky changes to our cells that lead to cancer. For example, some carcinogens can directly cause genetic mutations that foster abnormal cell growth and tumors. Furthermore, a carcinogen's link to cancer can depend on:

- Age and gender
- Potency: Some carcinogens require pretty heavy exposure to be dangerous, while others are linked to cancer with just a brief exposure.
- Exposure type: For example, were you exposed to a carcinogen one time or continually over a period of years?

Table 1: Carcinogens and types of Cancers

Agent	Cancer Type
Benzo [a]-pyrene (Tobacco)	Lung
Alcohol	Mouth, Pharynx, Larynx, Espohagus
Dietary Fat	Breast
Asbestos	Respiratory-tract, Pleural and Peritoneal Mesothelioma
Fermented Foods	Stomach
Estrogens	Endometrial, Ovarian, Breast
UV Light	Skin
X-Radiation and Gamma Radiation	Leukemia, Thyroid, Breast, Lung, Mouth, Stomach, Colon, Bladder, Ovarian, Skin, Central Nervous System
Tamoxifen	Endometrial
In-Utero Diethylstilbestrol	Childhood Cancer
Transabdominal Radiation	Childhood Cancer
Aflatoxin	Liver
Soot, Coal (Chimney Sweeping)	Scrotal
Nickel (Nickel Refining)	Lung, Nasal
Wood Dust (Woodworking)	Nasal
Cr(VI) (Leatherworking)	Lung
Mustard Gas	Respiratory-tract, Lung
2-naphthylamine	Bladder
HPV	Cervical, Oral, Pharyngeal, Vaginal, Vulvar, Head/Neck, Scrotal, Anal
H. pylori	Stomach
EBV	B-cell Lymphoma, Nasal, Pharyngeal, Stomach
Herpes Viruses	Kaposi's Sarcoma
Polyomaviruses (ex. JCV)	Brain
HTLV-1	T-cell Leukemia
Hepatitis B and C	Liver

Types of Carcinogens : Carcinogens can be grouped into one of three categories:

- Biologic carcinogens (e.g., types of bacteria)
- Chemical carcinogens (e.g., chemicals in tobacco smoke and asbestos).
- Physical carcinogens (e.g., UV radiation)

The term “physical carcinogen” encompasses multiple types of radiation (e.g., ultraviolet [UV] and ionizing radiation). Biologic carcinogens refer to viral and bacterial infections that

have been associated with cancer development (e.g., human papilloma virus [HPV] and hepatitis B virus [HBV]). Most carcinogens can be categorized as chemical carcinogens. In addition to heavy metals, organic combustion products, hormones, and fibers (e.g., asbestos), among others, are considered to be chemical carcinogens.

Biologic carcinogens:

Hepatitis B virus (HBV) and hepatitis C virus (HCV) are viruses that cause acute or chronic liver disease. They are known as “human carcinogens” because studies in humans show that chronic hepatitis B and hepatitis C infections cause liver cancer. Approximately one million United States residents are chronically infected with HBV, which primarily is transmitted through sexual contact (50%) and intravenous drug use (15%). HCV is the leading cause of liver disease in the United States with more than three million people infected. The major risk factor for hepatitis C infection is illegal intravenous drug use, which accounts for 60 percent of acute infections in adults. A vaccine is available for preventing hepatitis B infection but not hepatitis C infection. Infections can also be prevented by screening blood supplies, and by reducing contact with contaminated fluids in health care settings.

Human papilloma viruses (HPVs) are viruses that are sexually transmitted and can infect genital and mucous membranes. Some of these genital mucosal type HPVs are “known human carcinogens” because they cause cervical cancer in women. Approximately 20 million people in the United States are infected with genital HPVs, and 5.5 million new infections occur each year. Most people infected do not have symptoms, but some develop genital warts or cervical abnormalities.

Chemical carcinogens:

The induction of malignancy by a known or putative chemical carcinogen, which can be occupational (e.g., aromatic amines, arsenic, benzene, cadmium, chromium ores, soots, tars, vinyl chloride), environmental (e.g., aflatoxin, asbestos, tobacco) or iatrogenic (e.g., alkylating agents, anabolic steroids, phenacetin).

Initiation of the carcinogenic cascade occurs when an electrophilically reactive chemical (initiator) or, more often, one or more of its metabolites interacts with DNA, and repair of the damaged DNA is unsuccessful; this is followed by a sequence of events known as tumour promotion.

Understanding the relationship of chemicals to carcinogenesis has progressed significantly since the initial observations of Hill and Pott in the 1700's. Distinguishing between DNA-reactive chemicals and those which increase cancer risk by increasing cell proliferation has been a major breakthrough in delineating overall mechanisms. Competing processes for activation versus inactivation of chemicals occur at many levels, including metabolism, DNA repair, and cellular repair processes. These processes can be affected by other agents to decrease carcinogenesis (chemoprevention). Increasing knowledge of the multiple steps of carcinogenesis is leading to improved methods for screening chemicals for

carcinogenic activity and for regulatory decision making. Improvements in assessment of modes of action involved in animal and in vitro models have led to more rational approaches to assessing relevance to humans. The advent of genomics and high-throughput technologies have contributed to investigations of mechanisms and is beginning to impact development of better methods for screening chemicals.

Chemical exposure has been related to the development of cancer ever since the observation by John Hill, 1761 that snuff users developed nasal cancer more frequently than the general population. However, chemical carcinogenesis generally dates specifically to the observation by Sir Percival Pott in 1775 describing the frequent occurrence of cancer of the scrotum in chimney sweeps in England. He hypothesized that this was because of their significant exposure to soot. More importantly, he also proposed a mechanism to reduce the incidence of these cancers simply by requiring these individuals to bathe on a regular basis. This was instituted, and the incidence of scrotal cancer was essentially eliminated. It is important to note, given subsequent scientific discoveries and emphasis on extrapolation to low exposure levels, that he did not recommend eliminating the exposure completely, merely reducing it. Scrotal cancer today is a rare disease. Astute clinical observations such as that made by Pott, 1775 have been the basis for the discovery of many of the currently known classes of chemical carcinogens in humans. Examples include the observation by Rehn in 1895 that workers in the aniline dye industry in Germany frequently developed bladder cancer and more recent observations concerning the induction of angiosarcomas in patients exposed to contrast material used for radiologic imaging studies and vinyl chloride exposure in the workplace in Louisville, Kentucky. Research based on these observations led to several seminal discoveries in the history of chemical carcinogenesis. Investigation of coal tar, e.g., led to the first experimental induction of tumors in animal models by Yamagiwa and Ichikawa (1915) by painting this material on the skin of rabbits and mice. These models have been used in carcinogenesis research ever since. Similarly, the relationship of soot to cancer induction led to the purification and identification of the first pure chemical carcinogen by Kennaway and Hieger (1930) when they purified a small amount of dibenz[a,h]anthracene and produced tumors by painting the chemical on the backs of mice. The observation by Rehn (1895) of bladder cancer in the aniline dye industry led to the discovery by Kinoshita (1936) and Yoshida (1933) of the induction of liver cancer in rats by o-aminoazotoluene. This research also demonstrated the importance of dietary effects on the carcinogenic process. Ultimately, 2-naphthylamine was identified as one of the principle chemicals to which workers in the aniline dye industry were actually exposed, and Hueper et al. (1938) demonstrated that it was a bladder carcinogen when administered to dogs. In their study, they were also the first to emphasize the importance of latency. Their experiment involved administration of 2-naphthylamine to dogs for 2 years. The long latency period related to the development of cancer after chemical exposure has been an important consideration in theoretical models of carcinogenesis and in epidemiology ever since and has been a major barrier to the identification of additional chemicals and their possible relationship to

cancer. This was illustrated subsequently in numerous studies, such as demonstrating the relationship of cigarette smoking to lung cancer (and subsequently to other cancers), even though exposure frequently began during the teen years of the individuals, whereas lung cancers did not usually develop until after the age of 50 years. Even with high exposures to potent chemical carcinogens, such as 2-naphthylamine, benzidine, and vinyl chloride, the latency period is frequently 20–30 years or more.

Physical carcinogens

X-radiation and gamma-radiation:

X-radiation and gamma-radiation are “human carcinogens” because human studies show that exposure to these kinds of radiation causes many types of cancer including leukemia and cancers of the thyroid, breast and lung. The risk of developing cancers due to these forms of ionizing radiation depends to some extent on age at the time of exposure. Childhood exposure is linked to an increased risk for leukemia and thyroid cancer. Exposure during reproductive years increases the risk for breast cancer, and exposure later in life increases risk for lung cancer. Exposure to X-radiation and gamma radiation has also been shown to cause cancer of the salivary glands, stomach, colon, bladder, ovaries, central nervous system and skin. The total worldwide exposure to X-radiation and gamma-radiation, 55 percent is from low-dose medical diagnosis such as bone, chest and dental X-rays, and 43 percent is from natural sources like radon. Other sources, such as industry, scientific research, military weapons testing, nuclear accidents and nuclear power generation, account for about 2 percent.

Neutrons:

Neutrons cause genetic damage similar to that of X-radiation and gamma radiation, and thus can cause the same cancers. Neutron radiation is used less than other types of radiation in industry, medicine, and research. The general population is exposed to neutrons primarily from cosmic radiation that penetrates the earth’s atmosphere.

Naphthalene is used as an intermediate in the synthesis of many industrial chemicals, and has been used as an ingredient in some moth repellants and toilet bowl deodorants. Naphthalene is listed in the report as “reasonably anticipated to be a human carcinogen,” based on inhalation studies in animals which showed it causes rare nasal tumors in rats and benign lung tumors in female mice. MeIQ (2-Amino-3, 4-dimethylimidazo [4, 5-f]quinoline), MeIQx (2-Amino-3, 8-dimethylimidazo [4,5-f]quinoxaline), and PhIP (2-Amino-1-methyl-6-phenylimidazo [4,5-b]pyridine) are heterocyclic amine compounds formed when meats and eggs are cooked or grilled at high temperatures. These compounds are also found in cigarette smoke. They caused cancer in multiple organs including the forestomach, colon, liver, oral cavity, mammary gland, skin, and cecum. Several human studies suggest there is an increased risk for breast and colorectal cancers related to consumption of broiled or fried foods that may contain these or other similar compounds. Lead is used to make lead-acid storage batteries, ammunition, and cable coverings. Lead compounds are used in paint, glass

and ceramics, fuel additives, and in some ethnic and ceremonial cosmetics. The exposure to lead or lead compounds is associated with a small increased risk for lung or stomach cancer in humans, and cancer of the kidney, brain or lung in studies with laboratory animals. Cobalt Sulfate is used in electroplating, as coloring agents for ceramics, and as drying agents in inks and paints. Inhalation of Cobalt sulfate in laboratory animals causes adrenal gland and lung tumors. Diazoaminobenzene is a chemical used as an intermediate in the production of dyes and to promote adhesion of natural rubber to steel. It is metabolized to benzene, a “known human carcinogen,” and it causes genetic damage in laboratory animals. Nitrobenzene is a chemical used mainly in the production of other industrial chemicals. Inhalation of this compound produced cancer in experimental animals.

The carcinogens implicated as the main causative agents of the four most common cancers worldwide. These four cancers are lung, breast, colon, and stomach cancers. Together they account for about 41% of worldwide cancer incidence and 42% of cancer deaths.

Lung cancer : Lung cancer is the most common cancer in the world, both in terms of cases (1.6 million cases; 12.7% of total cancer cases) and deaths (1.4 million deaths; 18.2% of total cancer deaths). Lung cancer is largely caused by tobacco smoke. Risk estimates for lung cancer in the United States indicate that tobacco smoke is responsible for 90% of lung cancers. Other factors are implicated in lung cancer, and these factors can interact synergistically with smoking, so that total attributable risk adds up to more than 100%. These factors include occupational exposure to carcinogens (about 9-15%), radon (10%) and outdoor air pollution (1-2%). Tobacco smoke is a complex mixture of more than 5,300 identified chemicals. The most important carcinogens in tobacco smoke have been determined by a “Margin of Exposure” approach. Using this approach, the most important tumorigenic compounds in tobacco smoke were, in order of importance, acrolein, formaldehyde, acrylonitrile, 1,3-butadiene, cadmium, acetaldehyde, ethylene oxide and isoprene. Most of these compounds cause DNA damage by forming DNA adducts or by inducing other alterations in DNA. DNA damages are subject to error-prone DNA repair or can cause replication errors. Such errors in repair or replication can result in mutations in tumor suppressor genes or oncogenes leading to cancer.

Breast cancer: Breast cancer is the second most common cancer [(1.4 million cases, 10.9%), but ranks 5th as cause of death (458,000, 6.1%)]. Increased risk of breast cancer is associated with persistently elevated blood levels of estrogen. Estrogen appears to contribute to breast carcinogenesis by three processes;

- The metabolism of estrogen to genotoxic, mutagenic carcinogens
- The stimulation of tissue growth
- The repression of phase II detoxification enzymes that metabolize ROS leading to increased oxidative DNA damage. The major estrogen in humans, estradiol, can be metabolized to quinone derivatives that form adducts with DNA. These derivatives can cause depurination, the removal of bases from the phosphodiester backbone of DNA, followed by inaccurate repair or replication of the apurinic site leading to mutation and

eventually cancer. This genotoxic mechanism may interact in synergy with estrogen receptor-mediated, persistent cell proliferation to ultimately cause breast cancer. Genetic background, dietary practices and environmental factors also likely contribute to the incidence of DNA damage and breast cancer risk.

Colon cancer : Colorectal cancer is the third most common cancer. Tobacco smoke may be responsible for up to 20% of colorectal cancers in the United States. In addition, substantial evidence implicates bile acids as an important factor in colon cancer. Different studies indicate that the bile acids deoxycholic acid (DCA) and/or lithocholic acid (LCA) induce production of DNA damaging reactive oxygen species and/or reactive nitrogen species in human or animal colon cells. Furthermore 14 studies showed that DCA and LCA induce DNA damage in colon cells. Also 27 studies reported that bile acids cause programmed cell death (apoptosis). Increased apoptosis can result in selective survival of cells that are resistant to induction of apoptosis. Colon cells with reduced ability to undergo apoptosis in response to DNA damage would tend to accumulate mutations, and such cells may give rise to colon cancer. Epidemiologic studies have found that fecal bile acid concentrations are increased in populations with a high incidence of colon cancer. Dietary increases in total fat or saturated fat result in elevated DCA and LCA in feces and elevated exposure of the colon epithelium to these bile acids. When the bile acid DCA was added to the standard diet of wild-type mice invasive colon cancer was induced in 56% of the mice after 8 to 10 months. Overall, the available evidence indicates that DCA and LCA are centrally important DNA-damaging carcinogens in colon cancer.

Stomach cancer : Stomach cancer is the fourth most common cancer. *Helicobacter pylori* infection is the main causative factor in stomach cancer. Chronic gastritis (inflammation) caused by *H. pylori* is often long-standing if not treated. Infection of gastric epithelial cells with *H. pylori* results in increased production of reactive oxygen species (ROS). ROS cause oxidative DNA damage including the major base alteration 8-hydroxydeoxyguanosine (8-OHdG). 8-OHdG resulting from ROS is increased in chronic gastritis. The altered DNA base can cause errors during DNA replication that have mutagenic and carcinogenic potential. Thus *H. pylori*-induced ROS appear to be the major carcinogens in stomach cancer because they cause oxidative DNA damage leading to carcinogenic mutations.

Process of carcinogenesis

Cancer development is understood to be a multistep process. The concept of multi-stage carcinogenesis was first proposed by Berenblum and Schubik in 1948 and supported by later studies. Present day oncology recognizes three main phases: initiation, promotion and progression.

Initiation : The first stage of carcinogenesis has been labeled initiation since 1947. The conclusions reached from several experiments enabled the conclusion to be drawn that initiation is caused by irreversible genetic changes which predispose susceptible normal cells to malign evolution and immortality. The initiated cell is not a neoplastic cell but has taken

its first step towards this state, after successive genotypical and phenotypical changes have occurred. From a phenotypical perspective, the initiated cell is similar to the remaining cells. It undergoes mutations and these induce proliferation but not differentiation. Neoplasia initiation is essentially irreversible changes in appropriate target somatic cells. In the simplest terms, initiation involves one or more stable cellular changes arising spontaneously or induced by exposure to a carcinogen. This is considered to be the first step in carcinogenesis, where the cellular genome undergoes mutations, creating the potential for neoplastic development, which predisposes the affected cell and its progeny to subsequent neoplastic transformation. The human DNA sequences responsible for transformation are called oncogenes. At this stage, the initiated cells can remain latent for weeks, months or years, or they can grow in an autonomous and clonal fashion. This initiation process ensures that cellular division remains symmetrical by creating two new initiated cells. The clonal expansion of initiated cells results from a mitogenic process caused by an increased number of new cells and apoptosis inhibition, which prevents initiated cells from dying off. The increase in DNA damage is specifically important to stem cells, because they survive for a long time and exist in several tissues. In 1978, Potter explained that neoplastic cells could display a phenotype established between the embryonic aspect and the terminal differentiation, and that all neoplastic cells had monoclonal origin from a stem cell. Stem cells are immortal cells until they differentiate, or death is induced. If we delay their differentiation they become initiated and accumulate in tissues as clones of abnormal cells. Although stem cells are not identifiable in most tissues, it is believed that every tissue has a population of stem cells. Initiation is a fast, irreversible phenomenon and is transmitted to daughter cells. Cell proliferation is essential for this stage, if cellular division occurs before DNA repair systems can act then the injury becomes permanent and irreversible. Initiation is an additive process, neoplastic development depends on the carcinogenic dose, increasing the dose increases the incidence and the multiplicity of resultant neoplasias and reduces the latent period of its manifestation. Not all cells of a living organism exposed to an initiator agent will be initiated even if they have suffered mutations, and the genes that regulate the terminal differentiation must also be mutated.

Spontaneously initiated cells exist in all living organisms (Gomes-Carneiro et al. 1997, Trosko 2001). Initiation can begin with spontaneous mutations, supported by normal occurrences such as DNA depurination and deamination. Errors in DNA replication are also associated with initiation. Although spontaneous initiation is less common than induced initiation, its existence has been confirmed by the occurrence of spontaneous neoplasias in laboratory animals (Pitot and Dragan 1991, Gomes-Carneiro et al. 1997). Many of the active oncogenes have been isolated by molecular cloning, eg. Human bladder carcinoma, Burkitt's lymphoma, lung carcinoma, carcinoma of the breast and several others. Although the activation of more than one oncogene appears to be necessary for neoplastic transformation, the data imply that initiation may be induced with one hit kinetics. For example, in the human bladder carcinoma, a single point mutation converting the Ha-ras proto-oncogene into a potent oncogene was the first identified mutation in a human oncogene. Such tumor

gene mutations can have profound effects on cellular behavior and response, and can lead to dysregulation of genes involved in biochemical signaling pathways associated with control of cell proliferation and/or disruption of the natural processes of cellular communication, development and differentiation. However, the full expression of such neoplasia initiating mutations invariably requires interaction with other later arising gene mutations and/or changes to the cellular environment, but the initiating mutation creates the stable potential for pre-neoplastic cellular development in cells with proliferative capacity. The transformed cell undergoes continuous division with fidelity to the transformed karyotype and, possibly, with further mutations, before a malignant lesion is manifested.

Mechanisms of oncogene activation :

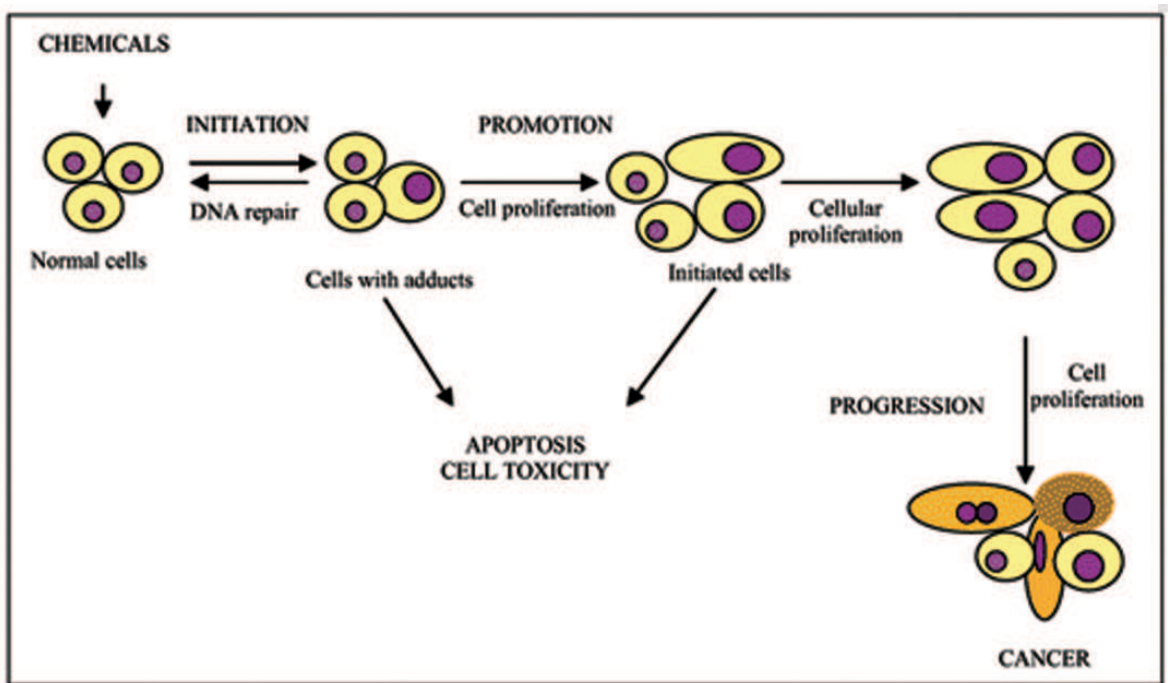


Fig: Chemical carcinogenesis stages and the occurrences involved in each one.

Each oncogene is closely associated with a normal DNA sequence present in the cellular genome, the proto-oncogene. At least five different mechanisms are considered for the conversion of proto-oncogenes to active oncogenes such as

- Over expression of proto-oncogene following acquisition of a novel transcriptional promoter. The oncogene then acquires activity because their transcripts are produced at much higher levels than those of the related normal proto-oncogene.
- Over-expression due to amplification of the proto-oncogene or oncogene. The increased gene copies cause corresponding increases in transcript and gene product.
- Influences on the levels of transcription and, in turn, the amount of gene product.
- Juxtaposition of the oncogene and immunoglobulin domains, following chromosomal

translocations, that appears to result in deregulation of the gene.

- Alteration in the structure of the oncogene protein. This is the best documented mechanism in the case of the oncogene proteins encoded by the ras genes.

The fourth and fifth mechanisms seem to be inter-related. A translocation can disturb the regulation of an oncogene by

- Providing a new promoter region or some other control element that would activate the oncogene; or
- Altering the coding sequence of a gene, changing its protein product from a benign to a malignant form.
- A close association between specific chromosomal translocations and certain human neoplasms has been demonstrated.

Promotion : The concept of promotion was introduced when chemical substances with low carcinogenic activity were discovered, which were still able to induce the development of cancer under experimental conditions. Promoter compounds do not interact directly with DNA and unchain biological effects without being metabolically activated. These agents increase cell proliferation in susceptible tissues, contribute towards fixing mutations, enhance alterations in genetic expression and cause changes in cellular growth control. On the other hand, these promoters may indirectly damage DNA by oxidation. At first, these occurrences were associated with epigenetic mechanisms, but nowadays it is widely agreed that promotion also involves genetic changes. Promoters delay the natural inhibition of the quiescent cells or in G0 by gap junctions. The promoters' most important activity is mitogenesis - genotoxic and mutational actions are not necessary at this stage. The promoter must be present for weeks, months and years in order to be effective and its effectiveness depends on its concentration in the target tissue. Promotion is a reversible stage, after a promoter's disappearance a regression in cell proliferation can occur, probably by apoptosis. It is stages that can be molded up by physiological factors and therefore limit the extent of experimental carcinogenesis. Some promoter agents are specific for a particular tissue, but others act simultaneously upon several tissues.

In studies of chemical carcinogenesis with prolonged exposure and using high doses almost all of the promoter agents induce neoplasias without initiation. Exposure to phenobarbital, benzene, asbestos, and arsenic even without the previous application of initiator agents leads to neoplastic development. This contradiction has two possible explanations: either the genotoxic effect was not identified by mutagenicity and genotoxicity assays, or the initiated cells emerged spontaneously. Not all cells exposed to promoters take part in the promotion stage, only cells which are stimulated to divide, that are undifferentiated, and have survived apoptosis, can contribute to instability between growth and cell death and lead to the appearance of a malign neoplasia. The transformed (initiated) cell can remain harmless,

unless and until it is stimulated to undergo further proliferation, upsetting the cellular balance. The subsequent changes of an initiated cell leading to neoplastic transformation may involve more than one step and requires repeated and prolonged exposures to promoting stimuli. Thus, in contrast to initiation which is induced at a rate of 0.1-1.0 per cell/Gy of radiation, the subsequent transforming event in the initiated cells occurs at a rate of only 10^{-6} to 10^{-7} per cell generation. Neoplastic development is influenced by the intra- and extracellular environment. Expression of the initial mutation will depend not only on interaction with other oncogenic mutations but also on factors that may temporarily change the patterns of specific gene expression, eg. cytokines, lipid metabolites, and certain phorbol esters. This may result in an enhancement of cellular growth potential and/or an uncoupling of the intercellular communication processes that restrict cellular autonomy and thereby coordinate tissue maintenance and development.

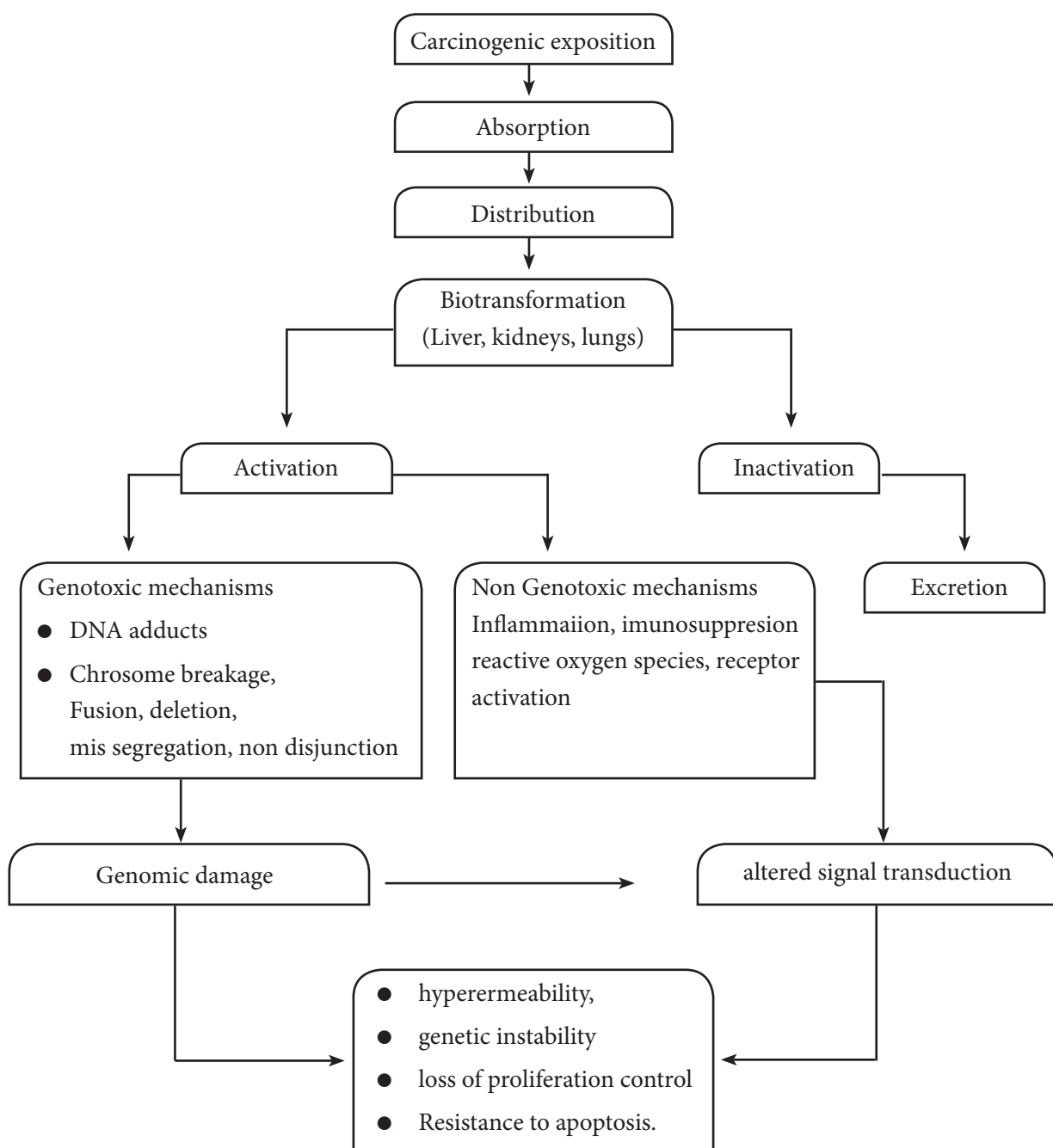
Progression: The sequence of lesions identified, via histopathology, between initiation and promotion are designated as preneoplastic lesions and/or benign neoplasias. Their transformation into malign lesions is the last stages of carcinogenesis and is the most extended - it is labeled progression. In progression, a neoplastic phenotype is acquired through genetic and epigenetic mechanisms. During progression, cell proliferation is independent from the presence of stimulus. Progression is characterized by irreversibility, genetic instability, faster growth, invasion, metastization and changes in the biochemical, metabolic and morphological characteristics of cells. Angiogenesis, as an epigenetic occurrence, is essential to neoplastic progression. The acquisition of an angiogenic phenotype precedes the development of characteristics that contribute to malignancy and its inhibition delays neoplastic development. It is the process through which successive changes in the neoplasm give rise to increasingly malignant sub-populations. Molecular mechanisms of tumor progression are not fully understood, but mutations and chromosomal aberrations are thought to be involved. The process may be accelerated by repeated exposures to carcinogenic stimuli or by selection pressures favoring the autonomous clonal derivatives. The initiated cells proliferate causing a fast increase in the tumor size. As the tumor grows in size, the cells may undergo further mutations, leading to increasing heterogeneity of the cell population. In the first phase of progression, sometimes referred to as neoplastic conversion, the pre-neoplastic cells are transformed to a state in which they are more committed to malignant development. This may involve further gene mutations accumulating within the expanding pre-neoplastic cell clone. The dynamic cellular heterogeneity, a feature of malignant development, may, in many instances, be a consequence of the early acquisition of gene specific mutations that destabilize the genome. Examples are mutations of the p53 gene or DNA mismatch repair genes.

Many tumor types develop transforming sequences in their DNA during their progression

from the normal to the cancerous state. An elevated mutation rate established relatively early in tumor development may, therefore, provide for the high-frequency generation of variant cells within a premalignant cell population. Such variant cells, having the capacity to evade the constraints that act to restrict proliferation of aberrant cells, will tend to be selected during tumorigenesis.

Absorption and metabolism of chemical carcinogens: Following exposure, chemical carcinogens may be absorbed in a number of ways (oral, inhalator, cutaneous, and injection) and distributed across several tissues. Absorption depends on the physicochemical properties of the substance and can take place via passive or active transport. The substances absorbed orally pass through the liver and only then are they distributed in the body; those absorbed in the lung are distributed by the blood before reaching the liver at a later stage. Those carcinogenic compounds classified as direct act directly on DNA, but most require enzymatic conversion and are thus labeled as indirect or procarcinogens. Metabolic activation is controlled by phase I reactions, while phase II reactions protect the body through the transformation of activated compounds into inert products which are easily eliminated from the body.

The performance of metabolic enzymes is essential for understanding chemical carcinogenesis and learning the differences between species as far as their susceptibility to neoplastic development is concerned. The enzymes in phase I participate in the reactions of oxidation, reduction and hydrolysis, and are classified as oxidoreductases (cytochrome P450 dependent monooxygenases, flavine monooxygenases, cyclooxygenases and alcohol dehydrogenase) and hydrolases (epoxide hydrolases). Phase II enzymes participate in the conjugation and inactivation of chemical carcinogens and include transferases (glutathione S-transferases, N-acetyltransferases, UDP-glucuronosyltransferases, sulphotransferases). Although these enzymes were originally only thought to be involved in the detoxification stages of biotransformation, they can also contribute to the activation of certain procarcinogens in vivo.



Metabolic activation occurs predominantly in the liver at the plain endoplasmic reticulum where the cytochrome P450 is more abundant, and to a lesser degree in the bladder, skin, gastrointestinal system, oesophagus, kidneys, and lungs. During this phase the cytochrome P450 mono-oxygenases introduces a reactive polar group into the carcinogenic, making it lipophylic. It then converts it into a powerful electrophilic product capable of establishing adducts with DNA. Phase II reactions are catalysed by hepatic and extra hepatic, cytoplasmic and cytochromic enzymes, acting separately or joined together. Conjugation reactions enable these enzymes to decompose the polar group in glucose, amino acids, glutathione

and sulphate, which are less toxic metabolites that are more soluble in water and more easily expelled by the urine and bile.

Peroxidations also occur parallel to metabolic reactions with the continuous production of reactive oxygen species (ROS). These radicals are associated with several chronic diseases including chemical carcinogenesis. The ROS damage DNA, RNA and proteins by chemical reactions such as oxidation, nitration/nitrosation and halogenations. This leads to an increase in mutations and alterations in the functions of important enzymes and proteins. Several experiments have proved that chemical compounds, which create ROS in excess, encourage initiation, promotion and neoplastic progression through genotoxicity. The impact of the ROS controlled by a cellular mechanism that operates at different levels: metabolism; reactions that maintain the redox balance in cells; transduction of the signal regulator of oxidation and DNA repair. The same enzyme may have the capacity to activate one chemical and deactivate another, all depending on its chemical structure. The specificity of the activation systems of different tissues regulate neoplastic development and is dependent on genetic polymorphism, which requires the expression and distribution of the enzymes involved in phase I and II reactions, and the resulting susceptibility to cancer development. People with a high quantity of phase I and a low quantity of phase II enzymes have a higher probability of synthesizing intermediate compounds and exhibiting more DNA damage. These have lead to incorrect interpretations when animal models are used in the research and analysis of carcinogenic properties of chemical compounds. Several studies have been developed in order to evaluate the differences between several exogenous and endogenous factors on individual susceptibility to carcinogenesis.

Tumor metastasis: As the tumor progression advances, the cells lose their adherence property, detach from the tumor mass and invade the neighboring tissues. The detached cells also enter the circulating blood and lymph and are transported to other organs/tissues away from the site of the primary growth and develop into secondary tumors at the new sites. These form the distant metastases, resulting in widely spread cancers. Cancer metastasis consists of a number of steps; the main steps are common for all tumors. The progress of the neoplastic disease depends on metastatic changes that facilitate:

- invasion of local normal tissues
- entry and transit of neoplastic cells in the blood and lymphatic systems
- the subsequent establishment of secondary tumor growth at distant sites.

Many of the steps in tumor metastasis involve cell-cell and cell-matrix interactions, involving specific cell surface molecules. Malignant cells are thought to have reduced ability to adhere to each other, so that they detach from the primary tumor and invade the surrounding tissues. The behavior of tumor is influenced by the cell adhesion molecules, one of the most important of which is cadherins. Animal studies have shown that a down-regulation of E-cadherin expression, resulting in lower levels, correlated with metastatic behavior in vivo, suggesting that cadherins function as invasion suppressor gene products. It is the metastatic

process and tumors spreading that are mainly responsible for the lethal effects of many common human tumors. In many cases gene mutations are believed to be the driving force for tumor metastasis, with the development of tumor vasculature playing an important role in the disease progression.

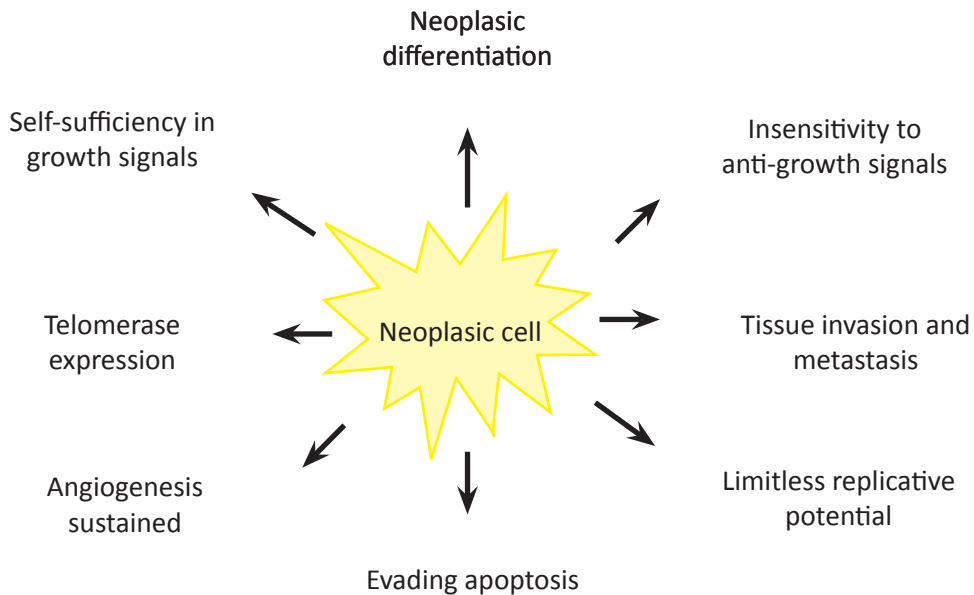


Fig: Malignant cell characteristics

Tumor angiogenesis : Tumor growth depends on the supply of growth factors and efficient removal of toxic molecules, which comes through an adequate blood supply. In solid tumors, efficient oxygen diffusion from capillaries occurs to a radius of 150-200(μ m), beyond which the cells become anoxic and die. Therefore, increase in tumor mass to more than 1-2 mm will depend on adequate blood supply through development of blood capillaries (angiogenesis). Schubik was the first to coin the term ‘tumor angiogenesis’ (Schubik, 1982). But it was Judah Folkman who hypothesized the importance of tumor angiogenesis in the development and metastasis of solid tumors. His theories are widely accepted today. Folkman and colleagues established that tumor growth beyond about 2 mm size could proceed only if a vascular supply is established. A number of tissue factors have been identified, which stimulate endothelial cell proliferation. These include the tumor angiogenesis factor (TAF), the vascular endothelial growth factor (VEGF), angioproteins - ang-1 and ang - 2, transforming growth factors (TGFs), interleukin - 1, and platelet-derived endothelial cell growth factor (PD-ECGF). Although the blood vessels that supply the developing tumors are derived from the host vasculature, their architecture differs considerably from that in the normal tissue. Tumor vessels are often dilated, saccular and tortuous and may contain tumor cells within the endothelial lining of the vessel. Therefore, the blood flow in the tumor may be sluggish

compared to that in the adjacent normal tissues and the tumor microvasculature may show hyperpermeability to plasma proteins.

Cancer genes : Somatic gene mutations are widely accepted as the basic event in the conversion of a normal cell into cancer cell. Many different genes are demonstrated to be involved in carcinogenesis. The gene mutation theory of oncogenesis maintains that carcinogens interact with DNA resulting in irreversible changes in the gene (point mutations), which predispose the cells to malignant transformation. The somatic genetic changes in cells that contribute to multistage tumor development potentially involve sequential mutation of different classes of genes, ie. Proto-oncogenes, tumor suppressor genes, genes involved in cell cycle regulation, and genes that play roles in maintaining normal genomic stability. Biochemical interactions between tumor gene mutations may destabilize the genome, compromise control of cell signaling, proliferation, and differentiation, and interfere with the normal interaction of cells in tissues. Two classes of regulatory genes are directly involved in carcinogenesis, the oncogenes and the antioncogenes.

Oncogenes : They are positive regulators of carcinogenesis. In non-transformed cells, they are inactive (proto-oncogenes). Gene mutations can activate proto-oncogenes, resulting in a gain of function. Several proto-oncogenes were first identified through viral transformation of cellular genome, eg. c-erbB, c-mos, c-myc, c-myb, C-H-ras. A large number of mutations in specific oncogenes - eg. ras, myc, etc have been found to be closely associated with different types of cancers.

Anti-oncogenes or tumor suppressor genes : They are negative growth regulators. Many human tumors, eg. retinoblastoma, Wilm's tumor, colon carcinoma, result from recessive mutation, which cause cancer when present on both homologues. These genes function as anti-oncogenes or tumor suppressor genes. In normal cells they regulate cell proliferation by checking cell cycle progression. Mutation in these genes results in a loss of gene function (the protein product will not be produced), which promotes carcinogenesis. Such gene mutations have been detected in several solid tumors, eg. cancers of breast, lung, rectum, etc., but only few such mutations have been seen in leukemias. The two most widely studied tumor suppressor genes are the Rb gene and p53 gene. The proteins encoded by these genes inhibit cell cycle progression by blocking transcription of gene products necessary for transition from G1 to S phase. Mutation in the Rb gene could lead to loss of normal inhibitory control of cell cycle progression and, thereby, increase cell proliferation. This effect, coupled with genetic changes that cause loss of apoptotic signals, would enhance malignant transformation. p53 has a major role in maintaining the genomic stability and cellular equilibrium. In normal cells, this gene promotes apoptosis, regulates cell cycle through G1 - S checkpoint control and induces cell differentiation. p53 participates in a cell cycle checkpoint signal transduction pathway that causes either a G1 arrest or apoptotic cell death after DNA damage. Mutations in p53, resulting in loss of function, will cause suppression of apoptosis, promote cell division by releasing the G1-S block and prevent differentiation of the cells,

leading to neoplasm development. Mutations in the p53 gene are the most common genetic change observed in a large number of human malignancies; at least 50% of all human cancers have been found to contain p53 abnormality. Mutations in this gene have been observed in a wide range of human cancers like cancers of the breast, lung, colon, skin, urinary bladder, ovary and lymphoid organs. More than 500 mutations of this gene have been documented in breast cancer.

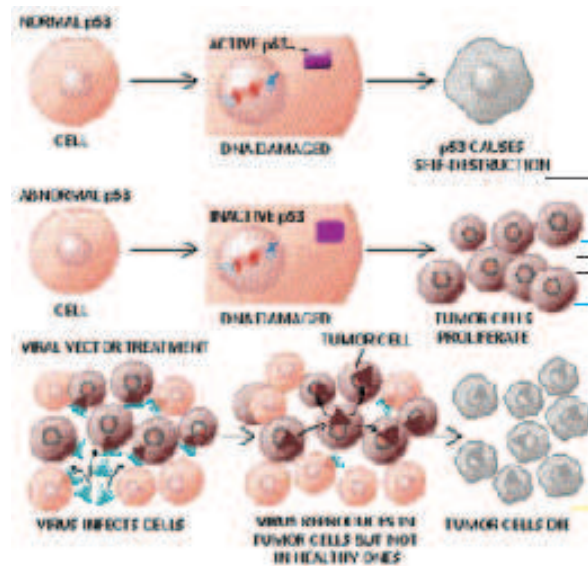


Fig: Role of p53 in the process of carcinogenesis.

Theories of carcinogenesis:

Gene mutation theory : This theory maintains that somatic gene mutations form the basis of neoplastic transformation and their clonal expansion leading to carcinogenesis. It is the most widely accepted and is supported by a large volume of experimental data. However, it does not explain tumor heterogeneity and aneuploidy and also the long latent periods between exposure to carcinogens and the development of tumors.

Aneuploidy theory : Another theory that is currently gaining momentum is the aneuploidy hypothesis. According to this hypothesis, a carcinogen initiates carcinogenesis by a preneoplastic aneuploidy, which destabilizes mitosis. This initiates an autocatalytic karyotype evolution that generates new chromosomal variants, including rare neoplastic aneuploidy. The aneuploidy hypothesis provides a plausible explanation for the long latent periods from carcinogen treatment to cancer development and the clonality.

Epigenetic theory : It has been recognized that non-mutational stable changes occur in cellular genome, which can contribute to carcinogenesis. Such events are broadly termed epigenetic and are thought to involve DNA methylation, genome imprinting and changes in DNA nucleoprotein structure. Increased levels of methylated cytosine (one of the pyrimidine bases in DNA) results in the elevation of spontaneous mutation rates in the affected genome.

While each theory has its own merits, it may not be possible to assign an exclusive role to a single process alone in carcinogenesis. In many cases, a combination of the two or all process may work in cooperation. An initiating somatic gene mutation can destabilize the genome and lead to aneuploidy and chromosome heterogeneity, characteristic of solid tumors, while epigenetic events can contribute to the neoplastic cell transformation and also facilitate promotional changes.

Cellular defense mechanisms in relation to cancer prevention and carcinogenesis:

Normal cells are naturally equipped with efficient defense mechanisms that work at different levels.

Antioxidants : The cells synthesize their own defense molecules, which include the non-protein thiol glutathione, and antioxidant enzymes like superoxide dismutase, catalase, glutathione peroxidase, reductase and S-transferases. These scavenge the ROS before they can reach the target molecules in the cell and thus protect against their attack on the vital molecules like DNA. Thus they serve as the biological watchdogs in safeguarding against free radical induced initiating changes, mutations and chromosomal aberrations. Many dietary ingredients like green vegetables, fruits, tea, spices and some diet supplements contain antioxidants. These include the vitamins A, C, and E, beta-carotene, alpha-tocopherol, ascorbic acid, flavonoids, lycopenes, curcumins and enzymes like caspase. They act as chemo-preventers by scavenging free radicals and enhancing cellular defense through their adaptogenic properties.

DNA repair : Damage to cellular DNA is the crucial early event in the neoplastic transformation of a cell. The DNA lesions may include altered bases, co-valent binding of bulky adducts, inter- and intra-strand crosslinks and generation of strand breaks. A range of alkylated products is formed in DNA by exposure to nitroso-compounds and other alkylating agents. Ionizing radiation and many genotoxic chemicals generate free radicals, which interact with DNA and produce different lesions ranging from base damage, deletions and complex and multiple lesions. Most normal cells possess a high capacity for repair of DNA damage. However, efficient repair depends on the type of damage, its severity and the time available for repair. The base damage and single strand breaks are repaired fast and without error, restoring the molecular structure. But double strand breaks and multiple breaks and local cluster lesions are not properly repaired and often contain errors (error-prone repair or misrepair), leading to cell death or cell survival with abnormal gene functions and chromosomal abnormalities which are associated with malignant cell transformation. DNA repair involves a number of genes, the products of which operate in a co-ordinated manner to form repair pathways that control restitution of DNA structure. Apoptosis or programmed cell death is an important mechanism of cellular defense in reducing the risks of error-prone repair. Cells with DNA damage undergo apoptosis, thus preventing these cells from surviving and entering the proliferating cell pool and, thereby, preventing the possibility of tumor development.

Apoptosis is a genetically controlled process involving p53, bcl2 and other genes. Mutations in p53 can block the tumor-suppressive effect by eliminating apoptosis, and thus, allowing the damaged cells to survive and undergo proliferation. Some of the gene products that control cell cycle also influence apoptotic tendencies, eg. c-myc, pRb, Tp53.

Role of diet in cancer control : Doll and Peto (1981) were the first to point out an association between dietary constituents and cancer. A vegetarian diet is considered to be beneficial in reducing cancer incidence. Epidemiological studies have suggested that a diet rich in vegetables and fruits reduces the risk of certain cancers. For example, diets rich in fibre, vitamins A, C, and E, beta-carotene, retinols, alpha-tocopherol, polyphenols, and flavonoids, and minerals like selenium and zinc, have cancer chemopreventive effect. Fruits and vegetables are rich sources of chemopreventive chemicals. These include inhibitors of carcinogen formation, blocking agents (block conversion of procarcinogens to carcinogens), stimulators of detoxifying system, trapping agents (trap and eliminate potential carcinogens) and suppressing agents (suppress the different steps of the metabolic pathway leading to cancer) (Stavric 1994). A study in China showed a high incidence of oesophageal and gastric cancers in a population whose diet is deficient in beta-carotene and vitamins C and E. An interventional program, where the diet was supplemented with beta-carotenes, vitamin E and selenium, produced a 20% reduction in the stomach cancer mortality over a period 5 years. WHO has recommended dietary intervention in the cancer control strategy for the new millennium. Dietary intervention follows two approaches:

- Intervention through supplementing with vitamins, antioxidants and other dietary factors.
- Intervention through dietary modification in which target levels are established for consumption of meat, fat, fiber, fruits and vegetables.

Cancer is a broad term to describe a large variety of diseases, the common feature of which is uncontrolled cell division. The process of carcinogenesis consists of three major steps: initiation, where an irreversible change is affected in the cellular genes; promotion, where the initiated cells expand by self-proliferation leading to abnormal growth and further mutations; and progression, where the cells detach from the primary tumor and invade other organs and tissues, forming metastatic growths. Angiogenesis plays an important role in the tumor metastasis. Different types of cancer genes - oncogenes and antioncogenes (tumor suppressor genes) - are involved in cancer development. Gain of function mutations in the oncogenes, leading to abnormal cell proliferation, and loss of function mutations in the anti-oncogenes leading to suppression of cell differentiation and apoptosis, are the major events leading to cancer development. Chromosomal aneuploidy and epigenetic events are also thought to be important. Several factors like age, sex, genetic predisposition, along with extrinsic factors like diet, environmental pollutants, alcoholism and tobacco habits have a major role in determining the cancer risk. Dietary intervention as a cancer preventive measure is a primary agenda on the WHO program.

CANCER AND DIFFERENT TYPES OF CANCER

Introduction

Cancer (medical term : malignant neoplasm) is a class of diseases in which a group of cells display uncontrolled growth (division beyond the normal limits), invasion (intrusion on and destruction of adjacent tissues), and sometimes metastasis (spread to other locations in the body via lymph or blood). These three malignant properties of cancers differentiate them from benign tumors, which are self-limited, do not invade or metastasize. Most cancers form a tumor but some, like leukemia, do not. The branch of medicine concerned with the study, diagnosis, treatment, and prevention of cancer is called oncology.

The body is made up of trillions of living cells. In the normal body, cells grow, divide to make new cells and die in an orderly way. During the early years of a person's life, normal cells divide faster to allow the person to grow. After the person becomes an adult, most cells divide only to replace worn-out or dying cells or to repair injuries. Cancer starts when cells in a part of the body start to grow out of control. Cancer cell growth is different from normal cell growth. Instead of dying, cancer cells continue to grow and form new, abnormal cells that the body doesn't need as well as invade (grow into) other tissues, something that normal cells can't do. Growing out of control and invading other tissues are what makes a cell a cancer cell because of DNA (deoxyribonucleic acid) damage. In a normal cell, when DNA is damaged the cell either repairs the damage or dies. In cancer cells, the damaged DNA is not repaired, but the cell doesn't die like it should. These new cells all have the same damaged DNA as the first abnormal cell does. People can inherit abnormal DNA (it's passed on from their parents), but most often DNA damage is caused by mistakes that happen while the normal cell is reproducing or by something in the environment. Sometimes the cause of the DNA damage may be cigarette smoking or sun exposure. But it's rare to know exactly what caused any one person's cancer. In most cases, the cancer cells form a tumor. Over time, the tumors can replace normal tissue, crowd it, or push it aside. Some cancers, like leukemia, rarely form tumors. Instead, these cancer cells involve the blood and blood-forming organs and circulate through other tissues where they grow. Cancer cells often travel to other parts of the body where they can grow and form new tumors. This happens when the cancer cells get into the body's bloodstream or lymph vessels. The process of cancer spreading is called metastasis. No matter where a cancer may spread, it's always named based on the place where it started. For example, colon cancer that has spread to the liver is called metastatic colon cancer, not liver cancer. In this case, cancer cells taken from the liver would be the same as those in the colon. They would be treated in the same ways too. Different types of cancer can behave very differently. For instance, lung cancer and skin cancer are very different diseases. They grow at different rates and respond to different treatments. This is why people with cancer need treatment that's aimed at their kind of cancer. Cancer may affect all animals,

people at all ages, even fetuses, but the risk for most varieties increases with age. Cancer causes about 13% of all deaths. According to the American Cancer Society, 7.6 million people died from cancer in the world during 2007. Nearly all cancers are caused by abnormalities in the genetic material of the transformed cells. These abnormalities may be due to the effects of carcinogens, such as tobacco smoke, radiation, chemicals, or infectious agents. Other cancer-promoting genetic abnormalities may be randomly acquired through errors in DNA replication, or are inherited, and thus present in all cells from birth. The heritability of cancers is usually affected by complex interactions between carcinogens and the host's genome. New aspects of the genetics of cancer pathogenesis, such as DNA methylation, and microRNAs are increasingly recognized as important.

Biological properties of cancer cells:

In 2000, Hanahan and Weinberg summarized the biological properties of malignant tumor cells as follows:

- Acquisition of self-sufficiency in growth signals, leading to unchecked growth.
- Loss of sensitivity to anti-growth signals, also leading to unchecked growth.
- Loss of capacity for apoptosis, in order to allow growth despite genetic errors and external anti-growth signals.
- Loss of capacity for senescence, leading to limitless replicative potential (immortality).
- Acquisition of sustained angiogenesis, allowing the tumor to grow beyond the limitations of passive nutrient diffusion.
- Acquisition of ability to invade neighboring tissues, the defining property of invasive carcinoma.
- Acquisition of ability to build metastases at distant sites, the classical property of malignant tumors (carcinomas or others).

The completion of these multiple steps would be a very rare event without loss of capacity to repair genetic errors, leading to an increased mutation rate (genomic instability), thus accelerating all the other changes. These biological changes are classical in carcinomas; other malignant tumor may not need all to achieve them all. For example, tissue invasion and displacement to distant sites are normal properties of leukocytes; these steps are not needed in the development of leukemia. The different steps do not necessarily represent individual mutations. For example, inactivation of a single gene, coding for the p53 protein, will cause genomic instability, evasion of apoptosis and increased angiogenesis. Not all the cancer cells are dividing. Rather, a subset of the cells in a tumor, called cancer stem cells, replicates them and generates differentiated cells.

The 6 hallmarks of cancer:

DNA mutations result in defects in the regulatory circuits of a cell, which disrupt normal cell proliferation behavior. However the complexity of this disease is not as simple at the

cellular and molecular level. Individual cell behavior is not autonomous, and it usually relies on external signals from surrounding cells in the tissue or microenvironment. There are more than 100 distinct types of cancers and any specific organ can contain tumors of more than one subtype. This provokes several questions. How many of these regulatory circuits need to be broken to transform a normal cell into a cancerous one? Is there a common regulatory circuit that is broken among different types of cancers? Which of these circuits are broken inside a cell and which of these are linked to external signals from neighboring cells in the tissue? The answer to these questions can be summarized in a heterotypic model, manifested as the six common changes in cell physiology that results in cancer (proposed by Douglas Hanahan and Robert Weinberg in 2000). This model looks at tumours as complex tissues, in which cancer cells recruit and use normal cells in order to enhance their own survival and proliferation. The 6 hallmarks of this currently accepted model can be described using a traffic light analogy

- ✓ Immortality: Continuous cell division and limitless replication
- ✓ Produce 'Go' signals (growth factors from oncogenes)
- ✓ Override 'Stop' signals (anti-growth signals from tumour suppressor genes)
- ✓ Resistance to cell death (apoptosis)
- ✓ Angiogenesis: Induction of new blood vessel growth
- ✓ Metastasis: Spread to other sites

Almost all cancers share some or all of the 6 traits depending on the tumour. Some tumours may show all these changes because of mutations in one key gene (e.g. the p53 gene controls at least 4 of the traits) whereas other tumours may need more than 1 mutation for progression. Genetic abnormalities found in cancer typically affect two general classes of genes. Cancer-promoting oncogenes are typically activated in cancer cells, giving those cells new properties, such as hyperactive growth and division, protection against programmed cell death, loss of respect for normal tissue boundaries, and the ability to become established in diverse tissue environments. Tumor suppressor genes are then inactivated in cancer cells, resulting in the loss of normal functions in those cells, such as accurate DNA replication, control over the cell cycle, orientation and adhesion within tissues and interaction with protective cells of the immune system. Diagnosis usually requires the histological examination of a tissue biopsy specimen by a pathologist, although the initial indication of malignancy can be symptoms or radiographic imaging abnormalities. Most cancers can be treated and some cured, depending on the specific type, location, and stage. Once diagnosed, cancer is usually treated with a combination of surgery, chemotherapy and radiotherapy. As research develops, treatments are becoming more specific for different varieties of cancer. There has been significant progress in the development of targeted therapy drugs that act specifically on detectable molecular abnormalities in certain tumors, and which minimize damage to normal cells. The prognosis of cancer patients is most influenced by the type of cancer, as well as the stage, or extent of the disease. In addition, histological grading and the presence of specific molecular markers can also be useful in establishing prognosis, as well as in

determining individual treatments. Cancer is generally classified according to the tissue from which the cancerous cells originate, the primary tumor, as well as the normal cell type they most resemble. These are location and histology, respectively. The following closely related terms may be used to designate abnormal growths:

Tumor : Originally, it meant any abnormal swelling, lump or mass. In current English, however, the word tumor has become synonymous with neoplasm, specifically solid neoplasm. Note that some neoplasms, such as leukemia, do not form tumors.

Neoplasm : The scientific term to describe an abnormal proliferation of genetically altered cells. Neoplasms can be benign or malignant: Malignant neoplasm or malignant tumor: synonymous with cancer. Benign neoplasm or benign tumor: a tumor (solid neoplasm) that stops growing by itself does not invade other tissues and does not form metastases. Invasive tumor is another synonym of cancer. The name refers to invasion of surrounding tissues. Pre-malignancy, pre-cancer or non-invasive tumor: A neoplasm that is not invasive but has the potential to progress to cancer (become invasive) if left untreated. These lesions are, in order of increasing potential for cancer, atypia, dysplasia and carcinoma in situ. The following terms can be used to describe a cancer:

Screening : A test done on healthy people to detect tumors before they become apparent. A mammogram is a screening test.

Diagnosis : The confirmation of the cancerous nature of a lump. This usually requires a biopsy or removal of the tumor by surgery, followed by examination by a pathologist.

Surgical excision : The removal of a tumor by a surgeon.

Surgical margins : The evaluation by a pathologist of the edges of the tissue removed by the surgeon to determine if the tumor was removed completely (“negative margins”) or if tumor was left behind (“positive margins”).

Grade : A number (usually on a scale of 3) established by a pathologist to describe the degree of resemblance of the tumor to the surrounding benign tissue.

Stage : A number (usually on a scale of 4) established by the oncologist to describe the degree of invasion of the body by the tumor.

Recurrence : New tumors that appear at the site of the original tumor after surgery.

Metastasis : New tumors that appear far from the original tumor.

Transformation : The concept that a low-grade tumor transforms to a high-grade tumor over time. Example: Richter’s transformation.

Chemotherapy : Treatment with drugs.

Radiation therapy : Treatment with radiations.

Adjuvant therapy : Treatment, either chemotherapy or radiation therapy, given after surgery to kill the remaining cancer cells.

Prognosis : The probability of cure after the therapy. It is usually expressed as a probability

of survival five years after diagnosis. Alternatively, it can be expressed as the number of years when 50% of the patients are still alive. Both numbers are derived from statistics accumulated with hundreds of similar patients to give a Kaplan-Meier curve. Cancers are classified by the type of cell that resembles the tumor and, therefore, the tissue presumed to be the origin of the tumor. Examples of general categories include:

Carcinoma : Malignant tumors derived from epithelial cells. This group represents the most common cancers, including the common forms of breast, prostate, lung and colon cancer.

Sarcoma : Malignant tumors derived from connective tissue, or mesenchymal cells.

Lymphoma and leukemia : Malignancies derived from hematopoietic (blood-forming) cells.

Germ cell tumor : Tumors derived from totipotent cells. In adults most often found in the testicle and ovary; in fetuses, babies, and young children most often found on the body midline, particularly at the tip of the tailbone; in horses most often found at the poll (base of the skull).

Blastic tumor or blastoma : A tumor (usually malignant) which resembles an immature or embryonic tissue. Many of these tumors are most common in children.

Malignant tumors (cancers) are usually named using carcinoma, sarcoma or blastoma as a suffix, with the Latin or Greek word for the organ of origin as the root. For instance, a cancer of the liver is called hepatocarcinoma; a cancer of the fat cells is called liposarcoma. For common cancers, the English organ name is used. For instance, the most common type of breast cancer is called ductal carcinoma of the breast or mammary ductal carcinoma. Here, the adjective ductal refers to the appearance of the cancer under the microscope, resembling normal breast ducts. Benign tumors (which are not cancers) are named using -oma as a suffix with the organ name as the root. For instance, a benign tumor of the smooth muscle of the uterus is called leiomyoma (the common name of this frequent tumor is fibroid). Unfortunately, some cancers also use the -oma suffix, examples being melanoma and seminoma.

Adult cancers : In the U.S. and other developed countries, cancer is presently responsible for about 25% of all deaths. On a yearly basis, 0.5% of the population is diagnosed with cancer. The statistics below are for adults in the United States, and may vary substantially in other countries. Most common (by occurrence) most common (by mortality) cancer is prostate cancer (33%), prostate cancer (10%), lung cancer (31%), lung cancer (12%), breast cancer (32%), breast cancer (15%), colorectal cancer (10%), colorectal cancer (10%), bladder cancer (7%), pancreatic cancer (5%), pancreatic cancer (6%), endometrial cancer (6%), ovarian cancer (6%), cutaneous melanoma (5%), leukemia (4%) and non-Hodgkin lymphoma (4%).

Child cancers : Cancer can also occur in young children and adolescents, but it is rare (about 150 cases per million yearly in the US). Statistics from the SEER program of the US NCI demonstrate that childhood cancers increased 19% between 1975 and 1990, mainly

due to an increased incidence in acute leukemia. Since 1990, incidence rates have decreased. Children living near nuclear facilities face an increased risk of cancer.

Infant cancers : The age of peak incidence of cancer in children occurs during the first year of life, in infants. The average annual incidence in the United States, 1975-1995 was 233 per million infants. Several estimates of incidence exist. According to SEER, in the United States neuroblastoma comprised 28% of infant cancer cases and was the most common malignancy among these young children (65 per million infants). The leukemia as a group (41 per million infants) represented the next most common type of cancer, comprising 17% of all cases. Central nervous system malignancies comprised 13% of infant cancer, with an average annual incidence rate of nearly 30 per million infants. The average annual incidence rates for malignant germ cell and malignant soft tissue tumors were essentially the same at 15 per million infants. Each comprised about 6% of infant cancer. According to another study leukemia (usually ALL) is the most common infant malignancy (30%), followed by the central nervous system cancers and neuroblastoma. The remainder consists of Wilms' tumor, lymphomas, rhabdomyosarcoma (arising from muscle), retinoblastoma, osteosarcoma and Ewing's sarcoma. Teratoma (a germ cell tumor) often is cited as the most common tumor in this age group, but most teratomas are surgically removed while still benign, hence not necessarily cancer. Prior to the widespread routine use of prenatal ultrasound examinations, the incidence of sacrococcygeal teratomas diagnosed at birth were 25 to 29 per million births. Female and male infants have essentially the same overall cancer incidence rates, a notable difference compared to older children. White infants have higher cancer rates than black infants. Leukemia's accounted for a substantial proportion of this difference: the average annual rate for white infants (48.7 per million) was 66% higher than for black infants (29.4 per million). Relative survival for infants is very good for neuroblastoma, Wilms' tumor and retinoblastoma, and fairly good (80%) for leukemia, but not for most other types of cancer.

Symptoms of cancer metastasis depends location of the tumor. Roughly, cancer symptoms can be divided into three groups:

Local symptoms : unusual lumps or swelling (tumor), hemorrhage (bleeding), pain and/or ulceration. Compression of surrounding tissues may cause symptoms such as jaundice (yellowing the eyes and skin).

Symptoms of metastasis (spreading) : enlarged lymph nodes, cough and hemoptysis, hepatomegaly (enlarged liver), bone pain, fracture of affected bones and neurological symptoms. Although advanced cancer may cause pain, it is often not the first symptom.

Systemic symptoms : weight loss, poor appetite, fatigue and cachexia (wasting), excessive sweating (night sweats), anemia and specific paraneoplastic phenomena, i.e. specific conditions that are due to an active cancer, such as thrombosis or hormonal changes.

Every symptom in the above can be caused by a variety of conditions (a list of which is referred to as the differential diagnosis). Cancer may be a common or uncommon cause of each item.

History of cancer :

The study of cancer, called oncology, is the work of countless doctors and scientists around the world whose discoveries in anatomy, physiology, chemistry, epidemiology and other related fields made oncology what it is today. Technological advances and the ever-increasing understanding of cancer make this field one of the most rapidly evolving areas of modern medicine. Cancer is the second leading cause of death in the United States. About one-half of all men and one-third of all women in the US will develop cancer during their lifetimes. Today, millions of people are living with cancer or have had cancer.

Human beings and other animals have had cancer throughout recorded history. So it's no surprise that from the dawn of history people have written about cancer. Some of the earliest evidence of cancer is found among fossilized bone tumors, human mummies in ancient Egypt, and ancient manuscripts. Growths suggestive of the bone cancer called osteosarcoma have been seen in mummies. Bony skull destruction as seen in cancer of the head and neck has been found, too. Oldest description of cancer (although the word cancer was not used) was discovered in Egypt and dates back to about 3000 BC. It's called the Edwin Smith Papyrus and is a copy of part of an ancient Egyptian textbook on trauma surgery. It describes 8 cases of tumors or ulcers of the breast that were removed by cauterization with a tool called the fire drill. The writing says about the disease, "There is no treatment". The origin of the word cancer is credited to the Greek physician Hippocrates (460-370 BC), described several kinds of cancer, referring to them with the Greek word *karkinos* (crab or crayfish) who is considered the "Father of Medicine." Hippocrates used the terms *carcinos* and *carcinoma* to describe non-ulcer forming and ulcer-forming tumors. This name comes from the appearance of the cut surface of a solid malignant tumour, with the veins stretched on all sides as the animal the crab has its feet, whence it derives its name. He later added the suffix -oma, Greek for swelling, giving the name carcinoma. Since it was against Greek tradition to open the body, Hippocrates only described and made drawings of outwardly visible tumors on the skin, nose, and breasts. Treatment was based on the humor theory of four bodily fluids (black and yellow bile, blood, and phlegm). According to the patient's humor, treatment consisted of diet, blood-letting, and/or laxatives. Through the centuries it was discovered that cancer could occur anywhere in the body, but humor-theory based treatment remained popular until the 19th century with the discovery of cells. In Greek, these words refer to a crab, most likely applied to the disease because the finger-like spreading projections from a cancer called to mind the shape of a crab. The Roman physician, Celsus (28-50 BC), later translated the Greek term into cancer, the Latin word for crab. Galen (130-200 AD), another Greek physician, used the word *oncos* (Greek for swelling) to describe tumors. Although the crab analogy of Hippocrates and Celsus is still used to describe malignant tumors, Galen's term is now used as a part of the name for cancer specialists – oncologists. Very early surgical treatment for cancer was described in the 1020s by Avicenna (Ibn Sina) in *The Canon of Medicine*. He stated that the excision should be radical and that all diseased tissue should be removed, which included the use of amputation or the removal of veins running in the

direction of the tumor. He also recommended the use of cauterization for the area being treated if necessary.

In the 16th and 17th centuries, it became more acceptable for doctors to dissect bodies to discover the cause of death. The German professor Wilhelm Fabry believed that breast cancer was caused by a milk clot in a mammary duct. The Dutch professor Francois de la Boe Sylvius, a follower of Descartes, believed that all disease was the outcome of chemical processes, and that acidic lymph fluid was the cause of cancer. His contemporary Nicolaes Tulp believed that cancer was a poison that slowly spreads, and concluded that it was contagious. With the widespread use of the microscope in the 18th century, it was discovered that the 'cancer poison' spread from the primary tumor through the lymph nodes to other sites ("metastasis"). This view of the disease was first formulated by the English surgeon Campbell De Morgan between 1871 and 1874. The use of surgery to treat cancer had poor results due to problems with hygiene. The renowned Scottish surgeon Alexander Monro saw only 2 breast tumor patients out of 60 surviving surgery for two years. In the 19th century, asepsis improved surgical hygiene and as the survival statistics went up, surgical removal of the tumor became the primary treatment for cancer. With the exception of William Coley who in the late 1800s felt that the rate of cure after surgery had been higher before asepsis (and who injected bacteria into tumors with mixed results), cancer treatment became dependent on the individual art of the surgeon at removing a tumor. During the same period, the idea that the body was made up of various tissues, that in turn were made up of millions of cells, laid rest the humoral theories about chemical imbalances in the body. The age of cellular pathology was born.

When Marie Curie and Pierre Curie discovered radiation at the end of the 19th century, they stumbled upon the first effective non-surgical cancer treatment. With radiation came also the first signs of multi-disciplinary approaches to cancer treatment. The surgeon was no longer operating in isolation, but worked together with hospital radiologists to help patients. The complications in communication this brought, along with the necessity of the patient's treatment in a hospital facility rather than at home, also created a parallel process of compiling patient data into hospital files, which in turn led to the first statistical patient studies.

Cancer patient treatment and studies were restricted to individual physicians' practices until World War II, when medical research centers discovered that there were large international differences in disease incidence. This insight drove national public health bodies to make it possible to compile health data across practices and hospitals, a process that many countries do today. The Japanese medical community observed that the bone marrow of bomb victims in Hiroshima and Nagasaki was completely destroyed. They concluded that diseased bone marrow could also be destroyed with radiation, and this led to the discovery of bone marrow transplants for leukemia. Since WWII, trends in cancer treatment are to improve on a micro-level the existing treatment methods, standardize them, and globalize them as a way to find cures through epidemiology and international partnerships.

Cancer in the sixteenth to eighteenth centuries :

During the Renaissance, beginning in the 15th century, scientists developed greater understanding of the human body. Scientists like Galileo and Newton began to use the scientific method, which later was used to study disease. Autopsies, done by Harvey (1628), led to an understanding of the circulation of blood through the heart and body that had until then been a mystery. In 1761, Giovanni Morgagni of Padua was the first to do something which has become routine today – he did autopsies to relate the patient's illness to pathologic findings after death. This laid the foundation for scientific oncology, the study of cancer. The famous Scottish surgeon John Hunter (1728-1793) suggested that some cancers might be cured by surgery and described how the surgeon might decide which cancers to operate on. If the tumor had not invaded nearby tissue and was “moveable,” he said, “There is no impropriety in removing it.” A century later the development of anesthesia allowed surgery to flourish and classic cancer operations such as the radical mastectomy were developed.

Cancer in the nineteenth century:

The 19th century saw the birth of scientific oncology with use of the modern microscope in studying diseased tissues. Rudolf Virchow, often called the founder of cellular pathology, provided the scientific basis for the modern pathologic study of cancer. As Morgagni had linked autopsy findings seen with the unaided eye with the clinical course of illness, so Virchow correlated microscopic pathology to illness. This method not only allowed a better understanding of the damage cancer had done, but also aided the development of cancer surgery. Body tissues removed by the surgeon could now be examined and a precise diagnosis could be made. The pathologist could also tell the surgeon whether the operation had completely removed the cancer.

Early theories about causes of cancer:

From the earliest times, physicians have puzzled over the causes of cancer. Ancient Egyptians blamed cancers on the gods.

Humoral theory : Hippocrates believed that the body had 4 humors (body fluids): blood, phlegm, yellow bile, and black bile. When the humors were balanced, a person was healthy. The belief was that too much or too little of any of the humors caused disease. An excess of black bile in various body sites was thought to cause cancer. This theory of cancer was passed on by the Romans and was embraced by the influential doctor Galen's medical teaching, which remained the unchallenged standard through the Middle Ages for over 1,300 years. During this period, the study of the body, including autopsies, was prohibited for religious reasons, which limited progress of medical knowledge.

Lymph theory : Among theories that replaced the humoral theory of cancer was the formation of cancer by another body fluid, lymph. Life was believed to consist of continuous and appropriate movement of the fluid parts of the body through the solid parts. Of all the fluids, the most important were blood and lymph. Stahl and Hoffman theorized that cancer

was composed of fermenting and degenerating lymph, varying in density, acidity, and alkalinity. The lymph theory gained rapid support. John Hunter, the Scottish surgeon from the 1700s, agreed that tumors grow from lymph constantly thrown out by the blood.

Blastema theory : In 1838, German pathologist Johannes Muller demonstrated that cancer is made up of cells and not lymph, but he believed that cancer cells did not come from normal cells. Muller proposed that cancer cells developed from budding elements (blastema) between normal tissues.

Chronic irritation theory : Virchow proposed that chronic irritation was the cause of cancer, but he believed incorrectly that cancers “spread like a liquid.” In the 1860s, German surgeon, Karl Thiersch, showed that cancers metastasize through the spread of malignant cells and not through some unidentified fluid.

Trauma theory : Despite advances in the understanding of cancer, from the late 1800s until the 1920s, trauma was thought by some to cause cancer. This belief was maintained despite the failure of injury to cause cancer in experimental animals.

Infectious disease theory : Two doctors in Holland, Zacutus Lusitani (1575-1642) and Nicholas Tulp (1593-1674) concluded at almost the same time that cancer was contagious. They made this conclusion based on their experiences with breast cancer in members of the same household. Lusitani and Tulp publicized the contagion theory in 1649 and 1652, respectively. They proposed that cancer patients should be isolated, preferably outside of cities and towns, in order to prevent the spread of cancer. Throughout the 17th and 18th centuries, some believed that cancer was contagious. In fact, the first cancer hospital in France was forced to move from the city in 1779 because people feared cancer would spread throughout the city. Although human cancer, itself, is not contagious, we now know that certain viruses, bacteria, and parasites can increase a person’s risk of developing cancer.

Development of modern knowledge about cancer:

Cancer is a diverse class of diseases which differ widely in their causes and biology. The common thread in all known cancers is the acquisition of abnormalities in the genetic material of the cancer cell and its progeny. Research into the pathogenesis of cancer can be divided into three broad areas of focus. The first area of research focuses on the agents and events which cause or facilitate genetic changes in cells destined to become cancer. Second, it is important to uncover the precise nature of the genetic damage, and the genes which are affected by it. The third focus is on the consequences of those genetic changes on the biology of the cell, both in generating the defining properties of a cancer cell, and in facilitating additional genetic events, leading to further progression of the cancer.

Mutation : chemical carcinogens

Cancer pathogenesis is traceable back to DNA mutations that impact cell growth and metastasis. Substances that cause DNA mutations are known as mutagens, and mutagens that cause cancers are known as carcinogens. Particular substances have been linked to specific

types of cancer. Tobacco smoking is associated with many forms of cancer, and causes 90% of lung cancer. Prolonged exposure to asbestos fibers is associated with mesothelioma. Many mutagens are also carcinogens, but some carcinogens are not mutagens. Alcohol is an example of a chemical carcinogen that is not a mutagen. Such chemicals may promote cancers through stimulating the rate of cell division. Faster rates of replication leaves less time for repair enzymes to repair damaged DNA during DNA replication, increasing the likelihood of a mutation. The incidence of lung cancer is highly correlated with smoking. Decades of research has demonstrated the link between tobacco use and cancer in the lung, larynx, head, neck, stomach, bladder, kidney, oesophagus and pancreas. Tobacco smoke contains over fifty known carcinogens, including nitrosamines and polycyclic aromatic hydrocarbons. Tobacco is responsible for about one in three of all cancer deaths in the developed world, and about one in five worldwide. Indeed, lung cancer death rates in the United States have mirrored smoking patterns, with increases in smoking followed by dramatic increases in lung cancer death rates and, more recently, decreases in smoking followed by decreases in lung cancer death rates in men. However, the numbers of smokers worldwide is still rising, leading to what some organizations have described as the tobacco epidemic.

Mutation : ionizing radiation : Sources of ionizing radiation, such as radon gas, can cause cancer. Prolonged exposure to ultraviolet radiation from the sun can lead to melanoma and other skin malignancies. Radio-frequency radiation from mobile phones has been proposed as a cause of cancer, but there is little evidence of such a link. Nevertheless, some experts caution against prolonged exposure.

Viral and chemical carcinogens : In 1915, Katsusaburo Yamagiwa and Koichi Ichikawa at Tokyo University, induced cancer in lab animals for the first time by applying coal tar to rabbit skin. More than 150 years had passed since clinician John Hill of London recognized tobacco as a carcinogen (a substance known or believed to cause cancer in humans). Many more years passed before tobacco was “rediscovered” as the most destructive source of chemical carcinogens known to man. Today we recognize and avoid many specific substances that cause cancer: coal tars and their derivatives (like benzene), some hydrocarbons, aniline (a substance used to make dyes), asbestos, and many others. Ionizing radiation from a variety of sources, including the sun, is also known to cause cancer. To ensure the public’s safety, the government has set safety standards for many substances, including benzene, asbestos, hydrocarbons in the air, arsenic in drinking water, and radiation. In 1911, Peyton Rous, at the Rockefeller Institute in New York, described a type of cancer (sarcoma) in chickens caused by what later became known as the Rous sarcoma virus. He was awarded the Nobel Prize for that work in 1968. Several viruses are now linked to cancer in humans, for example:

- Long-standing infection with the hepatitis B or C viruses can lead to cancer of the liver.
- One of the herpes viruses, the Epstein-Barr virus, causes infectious mononucleosis and has been linked to non-Hodgkin lymphomas and nasopharyngeal cancer.
- People with human immunodeficiency virus (HIV) have greater increased risk of

developing several cancers, especially Kaposi sarcoma and non-Hodgkin lymphoma.

- Human papilloma viruses (HPVs) have been linked to many cancers, especially those of the cervix, vulva, vagina, anus, and penis. Some head and neck cancers (mostly the tongue and tonsils) are linked to the high-risk types of HPV, too. Today there are vaccines to help prevent HPV infection.

The mode of virally-induced tumors can be divided into two, acutely-transforming or slowly-transforming. In acutely transforming viruses, the virus carries an overactive oncogene called viral-oncogene (v-onc), and the infected cell is transformed as soon as v-onc is expressed. In contrast, in slowly-transforming viruses, the virus genome inserts near a proto-oncogene in the host genome. The viral promoter or other transcription regulation elements then cause over expression of that proto-oncogene. This induces uncontrolled cell division. Because the site of insertion is not specific to proto-oncogenes and the chance of insertion near any proto-oncogene is low, slowly-transforming viruses will cause tumors much longer after infection than the acutely-transforming viruses. Hepatitis viruses, including hepatitis B and hepatitis C, can induce a chronic viral infection that leads to liver cancer in 0.47% of hepatitis B patients per year (especially in Asia, less so in North America), and in 1.4% of hepatitis C carriers per year. Liver cirrhosis, whether from chronic viral hepatitis infection or alcoholism, is associated with the development of liver cancer, and the combination of cirrhosis and viral hepatitis presents the highest risk of liver cancer development. Worldwide, liver cancer is one of the most common, and most deadly, cancers due to a huge burden of viral hepatitis transmission and disease.

In addition to viruses, researchers have noted a connection between bacteria and certain cancers. The most prominent example is the link between chronic infection of the wall of the stomach with *Helicobacter pylori* and gastric cancer. Although only a minority of those infected with *Helicobacter* go on to develop cancer, since this pathogen is quite common it is probably responsible for the majority of these cancers.

Age : Although cancer can occur in persons of every age, it is common among the aging population. Sixty percent of new cancer cases and two thirds of cancer deaths occur in persons > 65 years. The incidence of common cancers (eg, breast, colorectal, prostate, lung) increases with age. There are several theories as to why cancer incidence increases in the elderly: age-related alterations in the immune system (decreased immune surveillance); accumulation of random genetic mutations or lifetime carcinogen exposure (especially for colorectal and lung cancers); hormonal alterations or exposure; and long lifespan. Multiple genetic changes are necessary for the development of cancer, most clearly exemplified by the stepwise genetic changes shown by many colon polyps progressing to cancer. The exponential rise in many cancers with age fits with an increased susceptibility to the late stages of carcinogenesis by environmental exposures. Lifetime exposure to estrogen may lead to breast or uterine cancer; exposure to testosterone leads to prostate cancer. The decline in cellular immunity may also lead to certain types of cancer that are highly immunogenic (eg, lymphomas, melanomas).

Accumulation of DNA mutations have to be amplified to constitute a cancer, therefore the longer the lifespan, the higher the risk of developing cancer.

Hormonal imbalances : Some hormones can act in a similar manner to non-mutagenic carcinogens in that they may stimulate excessive cell growth. A well-established example is the role of hyperestrogenic states in promoting endometrial cancer.

Immune system dysfunction : HIV is associated with a number of malignancies, including Kaposi's sarcoma, non-Hodgkin's lymphoma, and HPV-associated malignancies such as anal cancer and cervical cancer. AIDS-defining illnesses have long included these diagnoses. The increased incidence of malignancies in HIV patients points to the breakdown of immune surveillance as a possible etiology of cancer. Certain other immune deficiency states (e.g. common variable immunodeficiency and IgA deficiency) are also associated with increased risk of malignancy.

Heredity : Most forms of cancer are "sporadic", and have no basis in heredity. There are, however, a number of recognized syndromes of cancer with a hereditary component, often a defective tumor suppressor allele. Famous examples are: Certain inherited mutations in the genes BRCA1 and BRCA2 are associated with an elevated risk of breast cancer and ovarian cancer. Tumors of various endocrine organs in multiple endocrine neoplasia (MEN types 1, 2a, 2b). Li-Fraumeni syndrome (various tumors such as osteosarcoma, breast cancer, soft tissue sarcoma, brain tumors) due to mutations of p53. Turcot syndrome (brain tumors and colonic polyposis) familial adenomatous polyposis an inherited mutation of the APC gene that leads to early onset of colon carcinoma. Hereditary nonpolyposis colorectal cancer (HNPCC, also known as Lynch syndrome) can include familial cases of colon cancer, uterine cancer, gastric cancer, and ovarian cancer, without a preponderance of colon polyps. Retinoblastoma, when occurring in young children, is due to a hereditary mutation in the retinoblastoma gene. Down syndrome patients, who have an extra chromosome 21, are known to develop malignancies such as leukemia and testicular cancer, though the reasons for this difference are not well understood.

Other causes : Excepting the rare transmissions that occur with pregnancies and only a marginal few organ donors, cancer is generally not a transmissible disease. The main reason for this is tissue graft rejection caused by MHC incompatibility. In humans and other vertebrates, the immune system uses MHC antigens to differentiate between "self" and "non-self" cells because these antigens are different from person to person. When non-self antigens are encountered, the immune system reacts against the appropriate cell. Such reactions may protect against tumour cell engraftment by eliminating implanted cells. In the United States, approximately 3,500 pregnant women have a malignancy annually, and transplacental transmission of acute leukaemia, lymphoma, melanoma and carcinoma from mother to fetus has been observed. The development of donor-derived tumors from organ transplants is exceedingly rare. The main cause of organ transplant associated tumors seems to be malignant melanoma that was undetected at the time of organ harvest. A few types

of cancer in non-humans have been found to be caused by transmission of the tumor cells themselves. This phenomenon is seen in dogs with Sticker's sarcoma, also known as canine transmissible venereal tumor, as well as Devil facial tumour disease in Tasmanian devils.

As of 2014, the World Health Organization's International Agency for Research on Cancer (IARC) has identified more than 100 chemical, physical, and biological carcinogens. Many of these associations were recognized long before scientists understood much about how cancer develops. Today, research is discovering new carcinogens, explaining how they cause cancer, and providing insight into ways to prevent cancer. By the middle of the 20th century, scientists had the instruments they needed to work on some of the complex problems of chemistry and biology that remained unsolved. James Watson and Francis Crick, who received a Nobel Prize in 1962 for their work, had discovered the exact chemical structure of DNA, the basic material in genes. DNA was found to be the basis of the genetic code that gives orders to all cells. After learning how to translate this code, scientists were able to understand how genes worked and how they could be damaged by mutations (changes or mistakes in genes). These modern techniques of chemistry and biology answered many complex questions about cancer. Scientists already knew that cancer could be caused by chemicals, radiation, and viruses, and that sometimes cancer seemed to run in families. But as the understanding of DNA and genes increased, they learned that it was the damage to DNA by chemicals and radiation, or the introduction of new DNA sequences by viruses that often led to the development of cancer. It became possible to pinpoint the exact site of the damage on a specific gene. Scientists discovered that sometimes defective genes are inherited, and sometimes these inherited genes are defective at the points where certain chemicals also tend to cause damage. In other words, most of the things that caused cancer (carcinogens) caused genetic damage (mutations) that looked a lot like the mutations that could be inherited and could result in the same types of cancer if more mutations were introduced. No matter which way the first mutation started (inborn or spontaneous), the cells that grew from the mutated cells led to groups of abnormal cells (called clones, or duplicates of the abnormal cell). The mutant clones evolved to even more malignant clones over time, and the cancer progressed by more and more genetic damage and mutations. The big difference between normal tissues and cancer is that normal cells with damaged DNA die, while cancer cells with damaged DNA do not. The discovery of this critical difference answered many questions that had troubled scientists for many years.

Molecular causes of cancer :

Evading growth suppressors :

Cell proliferation in normal cells is a tightly controlled process wherein the pro-and antiproliferation signals coordinate their activities at the cell cycle level. Particularly, the G1 phase of the cell cycle is a vital checkpoint wherein the antigrowth signals exert their influence to block cell proliferation. Antigrowth signals in normal cells can block proliferation in multiple ways:

- Induction of the G0 phase
- Induction of a postmitotic state, usually involving terminal differentiation of the cell.
- However, most cancer cells circumvent normal growth suppressors in order to continue proliferating.

The tumor suppressors most commonly dysregulated in cancer cells are retinoblastoma protein (Rb) and p53. In normal tissue, these proteins are part of a large network that controls the cell cycle. Rb actively inhibits cell passage through the restriction point in the G1 cell cycle phase. Cancer cells with mutated Rb remove this gatekeeper and allow for ongoing cell proliferation. p53 functions as a central regulator of apoptosis because it arrests the cell cycle in cells with DNA damage. Loss of p53 allows for cell cycle progression despite DNA damage and cellular stresses. Rb and p53 are common tumor suppressors that are inactivated in tumor cells, leading to uncontrolled growth and proliferation.

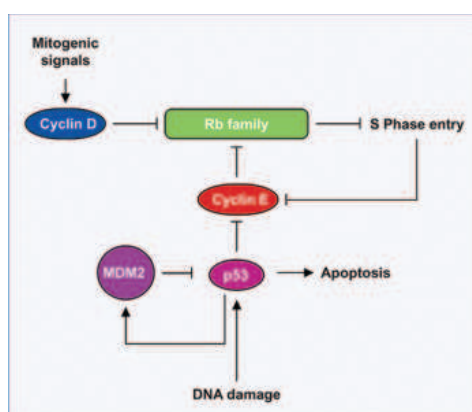


Fig: Evading growth suppressors

Emerging hallmark: evading immune destruction :

Immune surveillance is an essential cellular process that proactively prevents tumor formation in the human body. Preclinical studies have suggested that an active immune system continuously recognizes and eliminates the vast majority of cancer cells before they establish themselves and form a tumor mass. However, cancer immunoediting, an emerging hallmark, includes 3 key phases—elimination, equilibrium and escape.

- The immune system successfully recognizes and eliminates cancer cells, a process often described as the elimination phase.
- Tumor cells not eliminated by the immune system proceed to the equilibrium phase, in which the immune system controls cancer cell growth but does not completely eliminate the transformed cells.
- Tumor cells not susceptible to immune destruction progress into the escape phase. In this phase, the “escaped” tumor clones—not effectively detected and destroyed by the immune system—continue to divide and grow.

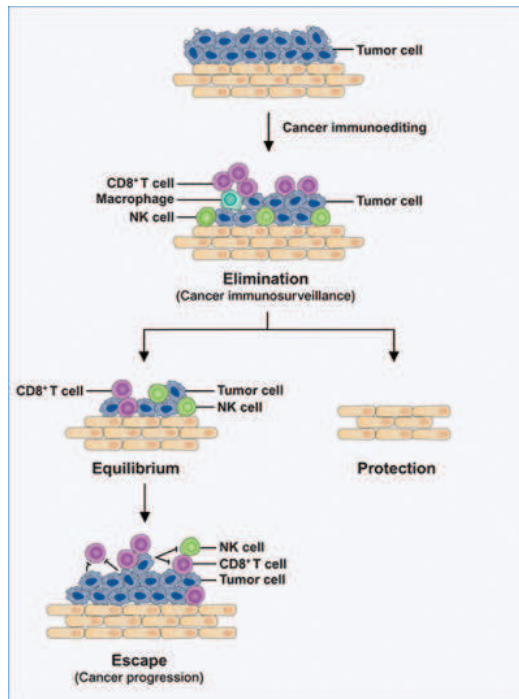


Fig: evading immune destruction

Clinical examples also support this finding, demonstrating that colorectal and ovarian cancer patients with an increased immune response have a better prognosis than do those patients with a reduced immune response. Cancer cells that successfully navigate these phases acquire the ability to evade immune destruction.

Sustaining proliferative signaling :

Growth signaling in normal cells is a highly regulated process wherein proliferative signals are activated whenever necessary and deactivated when no longer necessary; this tight regulation ensures cell homeostasis. However, in cancer cells this regulation is compromised. One of the fundamental traits of cancer cells is their ability to proliferate without a controlled signaling input. They achieve this in a number of ways:

- Increasing growth factor production
- Stimulating normal cells in the microenvironment to provide cancer cells with growth factors
- Increasing the number of receptors on the cell surface
- Structurally altering receptors to facilitate cancer cell signaling
- Activating proteins in the downstream signaling pathway.

Recent studies also highlight the ability of cancer cells to disrupt negative feedback loops that constitute a safety mechanism to dampen a signaling pathway whenever a mitogenic signal is hyperactivated. One key example of this is the RAS oncoprotein. Oncogenic activity

of RAS is not the result of overactive RAS signaling but rather the disruption of normal negative feedback mechanisms operated by the oncogenic GTPase. Other examples of this process include loss-of-function mutations in phosphatase and tensin homolog (PTEN), which amplify hosphatidylinositol 3-kinase (PI3K) signaling. Tumor cells disrupt negative feedback loops in the oncogenic RAS signaling pathway, leading to sustained proliferative signaling in tumor cells.

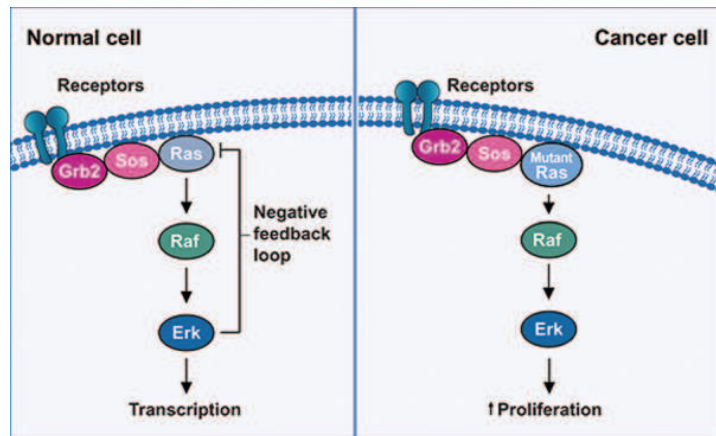


Fig: Sustaining proliferative signaling

Enabling replicative immortality:

Normal cells have a finite replicative ability. An intrinsic cellular mechanism allows normal cells to divide a finite number of times and blocks cell division beyond a certain limit. Cancer cells overcome this by over expressing telomerase, an enzyme that maintains telomere length, which protects the ends of chromosomes and allows the cell to continue proliferating. This process is also aided in part by the loss of tumor suppressor genes such as p53. In recent years, molecular cancer research has uncovered additional functions of telomerase that are independent of telomere maintenance and may aid in tumor growth:

- Enhancement of cell proliferation and/or resistance to apoptosis
- DNA damage repair
- RNA-dependent RNA polymerase function
- Association with chromatin

A shortening of telomere length activates replicative senescence in normal cells; however, tumor cells overcome the finite replicative ability by over expressing telomerase, an enzyme that maintains telomere length.

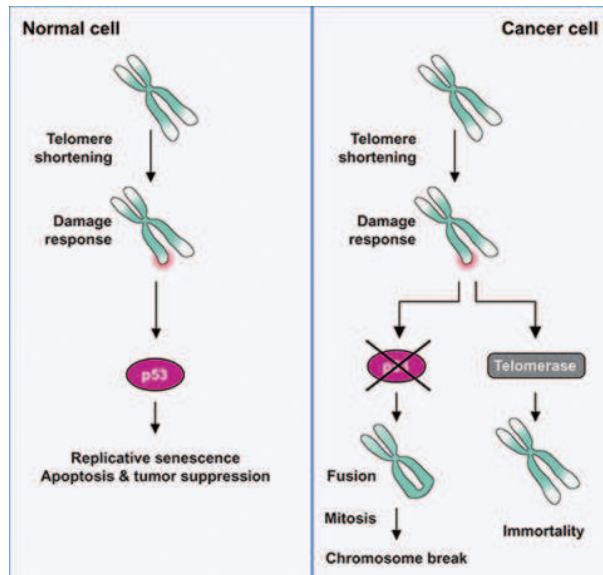


Fig: Enabling replicative immortality

Enabling characteristic: genome instability and mutation :

Multiple alterations in the genomes of cancer cells serve as the foundation for many oncogenic processes. Cancer cells take advantage of increased rates of mutations in order to accumulate several mutations needed to foster tumorigenesis. They do this through

- Increased sensitivity to mutagenic agents
- Breakdown one or more of the cell's DNA repair mechanisms mediated by genes such as p53 or breast cancer type 1 susceptibility protein (BRCA1)
- A combination of these factors.

Accumulation of these mutations is accelerated by altering DNA-maintenance machinery, or “caretaker” genes. These genes are responsible for

- Detecting DNA damage and activating repair machinery
- Directly repairing damaged DNA
- Inactivating or intercepting mutagenic molecules

By inactivating or suppressing caretaker genes, tumor cells can increase the rate of mutations and, subsequently, tumorigenesis. Analyses of cancer cell genomes also reveal function-altering mutations and demonstrate that genomic instability increases during tumor progression. Cancer cells take advantage of mutations in DNA repair pathways to promote genomic instability. Depicted above is one such mechanism, resulting from the defective BRCA signaling pathway.

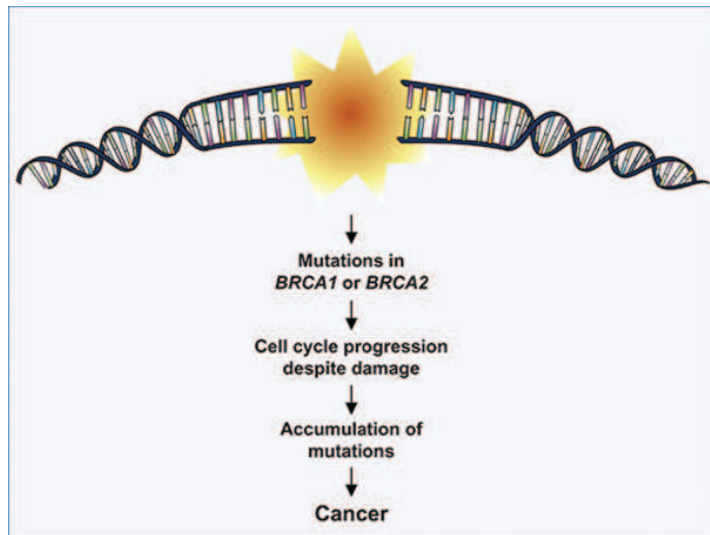


Fig: Genome instability and mutation

Activating invasion and metastasis :

Tissue invasion and metastasis are integral components in how tumor cells escape from the primary site and disseminate into distant organs. The process of tissue invasion and metastasis is not well understood, but, in general, it involves changes in the way cells attach to other cells and to the extracellular matrix. This process has several steps, including

- Local tissue invasion
- Intravasation
- Transition through the blood and lymphatic system
- Colonization of foreign tissue

Molecular cross-talk between tumor cells and neoplastic stroma suggests that metastases do not arise from a cell-autonomous model but require input from surrounding tissue. One example of this is the involvement of tumor-associated macrophages (TAMs) that supply cancer cells with epidermal growth factor (EGF) and colony-stimulating factor 1 (CSF-1) and assist with intravasation. Molecular cancer research into the complexity of metastatic growth also shows that different malignancies exhibit different characteristics. Distinct modes of invasion are seen in metastatic and nonmetastatic diseases. The reasons for this remain elusive, but they may be due to different cell-biological programs. Genetic pathways, such as that of tumor necrosis factor α (TNF- α) in bone dissemination, may facilitate tumor metastasis to preferred organ destinations. Tumor cell migration is promoted in part through a paracrine loop involving CSF-1, EGF, and their corresponding receptors, which are differentially expressed on carcinoma cells and macrophages residing in the tumor microenvironment.

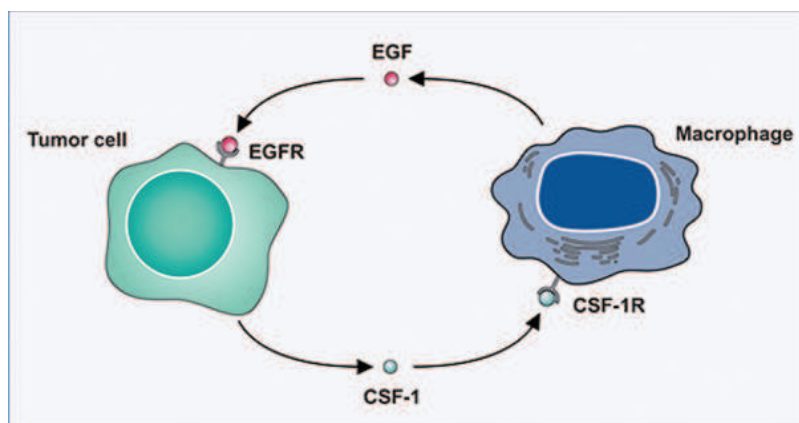


Fig: Activating invasion and metastasis

Enabling characteristic: tumor-promoting inflammation

The tumor microenvironment is often infiltrated by innate and adaptive immune system cells that enable tumors to mimic inflammatory conditions seen in normal tissues. Current molecular cancer research indicates that this tumor-associated inflammation might aid in tumor growth. Emerging research also indicates that tumor-associated inflammation may aid in tumor growth by supplying the tumor microenvironment with

- Growth factors
- Survival factors
- Pro-angiogenic factors

Extracellular matrix (ECM)–modifying enzymes that promote angiogenesis, invasion, and metastasis. Inductive signals that activate epithelial-mesenchymal transition (EMT) and other hallmark-facilitating mechanisms. Additionally, inflammation is often seen in early stages of neoplastic disease. Early inflammation can release chemicals into the tumor microenvironment and may lead to genetic mutations that enable and accelerate the formation of a tumor. Tumor-associated inflammation may promote tumor growth by supplying the microenvironment with growth factors, survival factors, and factors that promote angiogenesis.

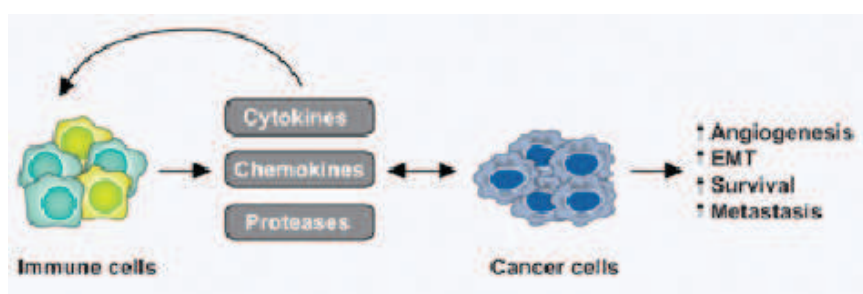


Fig: tumor-promoting and inflammation

Inducing angiogenesis:

In tumor cells, the process of angiogenesis, or the formation of new blood vessels, is critical for sustained tumor growth and metastasis. Tumor angiogenesis is a multistep process and involves signaling input from several pro-angiogenic growth factors. The moment at which a tumor begins to overexpress pro-angiogenic factors, such as vascular endothelial growth factor (VEGF), is generally referred to as the “angiogenic switch. Unabated angiogenesis enables tumor expansion and local invasion through

- Delivery of oxygen and nutrients
- Production of growth factors that benefit tumor cells.

Molecular cancer research also suggests that metastases can ultimately exit through the new tumor vasculature into systemic circulation. Two additional components play a role in tumor neovascularization

- Pericytes are supporting cells that have long been associated with normal tissue vasculature; however, recent studies reveal that pericyte coverage is also important for tumor angiogenesis.
- Molecular cancer research also indicates that bone marrow–derived cells, such as macrophages and neutrophils, are recruited to lesions and may help initiate the angiogenic switch. Tumor angiogenesis is a function of multiple signals from a number of cell types residing in the tumor microenvironment.

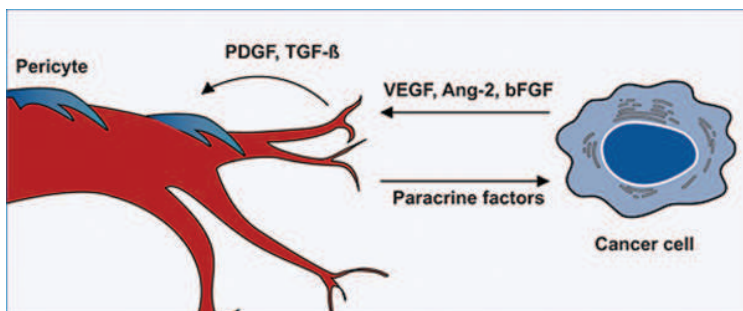


Fig: Inducing angiogenesis:

Resisting cell death :

Normal cells switch on apoptosis in response to stress-inducing events such as irreparable DNA damage. However, cancer cells in general avoid invoking a cell death response when challenged with a cellular stress. Virtually all cancer cells dysregulate signaling pathways by over expressing anti-apoptotic proteins and silencing pro-apoptotic proteins. The extrinsic pathway is activated when pro-apoptotic ligands bind to pro-apoptotic cell surface receptors. The intrinsic pathway is triggered from within the cell by developmental cues or severe cell stress, such as DNA damage.

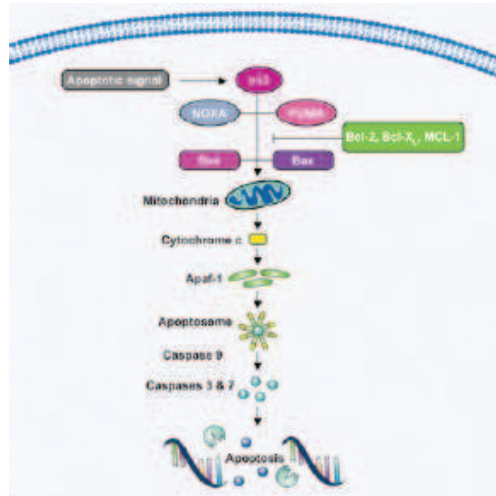


Fig: Resisting cell death.

This pathway is more widely implicated in tumorigenesis. Cancer cells also resist cell death by altering normal cellular autophagy and necrosis. Autophagy, or the process of breaking down organelles for energy metabolism, is part of overall cell regulation and may be inactivated in cancer cells. Necrosis, or cell death, releases pro-inflammatory signals to the surrounding microenvironment and may actively promote tumor growth. The intrinsic apoptotic signaling pathway is activated as a safety mechanism in response to severe cell stress, such as DNA damage. Tumor cells accumulate genetic alterations that disable this vital apoptotic-signaling arm.

Identification and histopathology of cancers:

The most common techniques for detecting cancer are imaging techniques such as MRI, X-rays (such as mammograms), CT, and ultrasound, which can provide an image of a tumor. Endoscopy allows a physician to insert a lighted instrument to look for tumors in organs such as the stomach, colon, and lungs. Most of these techniques are used to detect visible tumors, which must then be removed by biopsy and examined microscopically by a pathologist. The pathologist looks for abnormalities in the cells in terms of their shape, size, and structure, especially the nucleus. In addition, the pathologist looks at the borders of the tumor to see whether those cells are normal. Based on examination of the tumor cells, the pathologist determines whether the tumor is benign or malignant, and determines whether it is in an early or late stage of development. Diagnosis may also include the removal and examination of lymph nodes to determine whether the cancer cells have spread. Tumor markers are proteins found more often in the blood of individuals with the tumor than in normal individuals. These are not ideal compounds for diagnosing cancer for two reasons. First, individuals without cancer may have elevated levels of the marker, leading to false positives. Second, tumor markers are not sufficiently elevated in all individuals with cancer to allow their detection. This leads to false negatives. One of the most commonly used tumor markers is prostate-

specific antigen (PSA). It is present in all adult males, but its level is increased after both benign and malignant changes in the prostate. Therefore, high levels of PSA indicate only that further tests are required to determine whether the condition is cancer. If prostate cancer is diagnosed, the levels of PSA can help to determine the effectiveness of treatment and detect recurrence. Another tumor marker is CA125, which is produced by a number of different cells, particularly ovarian cancer cells. It is used primarily to monitor the treatment efficacy of ovarian cancer. When the cancer is responding to treatment, CA125 levels fall. It is not used as a routine test for ovarian cancer because many common conditions that cause inflammation also increase the level of CA125, leading to a high incidence of false positives. The earlier a cancer is found the more effectively it can be treated; however, early stage cancers typically produce no symptoms. Scientists are developing molecular techniques to detect very early cancer. Using techniques such as mass spectrometry, they are also developing specific blood tests to identify a pattern of new proteins in the blood of individuals with a particular type of cancer. In addition, scientists are developing DNA microarrays to identify genes expressed in particular types of cancer cells. With the sequencing of the human genome and the mapping of single nucleotide polymorphisms (SNPs), it may be possible to diagnose particular cancers by identifying cells with known gene alterations. In 2002 scientists detected ovarian cancer by testing blood for the presence of DNA released by tumor cells. They looked for changes in certain alleles at eight SNPs that are characteristic of cancer. Using this technique, they successfully identified eighty-seven percent of patients known to have early-stage of ovarian cancer and ninety-five percent of those with late-stage ovarian cancer. The ability to determine which genetic alterations are associated with various cancers opens up the possibility of identifying cancerous cells while the cancer is in an early, treatable stage.

There are several benefits to identifying and classifying cancers using histological sections and staining methodology.

- **Diagnosis :** Microscopic observation helps determine whether the tumour tissue is benign (harmless) or malignant (potentially fatal). Gross cellular morphology and tissue specific markers are used to classify cancerous cells.
- **Therapy :** The tissue diagnosis given by the pathologist indicates the type of cell that is proliferating, its histological grade, genetic abnormalities, and other features of the tumor. Together, this information is useful to evaluate the prognosis of the patient and to choose the best treatment. Cytogenetics and immunohistochemistry are other types of testing that the pathologist may perform on the tissue specimen. These tests may provide information about the molecular changes (such as mutations, fusion genes, and numerical chromosome changes) that has happened in the cancer cells, and may thus also indicate the future behavior of the cancer (prognosis) and best treatment. Cancer progression can be predicted by histotyping, e.g. patients with simple hyperplasia in the uterine epithelium have <1% chance of developing cancer compared 82% risk in patients with atypical hyperplasia.

- **Cellular origin (histogenesis)** : Determining the origins of the tumour by histopathological classification of tissue is useful in identifying whether the tumour is a primary or secondary tumour e.g. a liver tumour may have metastasized from elsewhere or source of origin of the tumour e.g. lung cancer due to smoking is epithelial (lung carcinoma) but due to asbestos exposure is mesothelial or asbestos cancer.

Oncogenes and tumor suppressor genes

During the 1970s, scientists discovered 2 particularly important families of genes related to cancer: oncogenes and tumor suppressor genes.

Oncogenes : An oncogene is a gene that encodes a protein capable of transforming cells in culture or inducing cancer in animals. These oncogenes are the derivatives of normal cellular genes called proto-oncogenes. Proto-oncogenes code for proteins that stimulate cell cycle division but mutated forms, called oncogenes, cause stimulatory proteins to be overactive, with the result that cells proliferate excessively. Oncogenes are dominant over proto-oncogene. No cell can survive in isolation. Every cell is part of a community, which forms a tissue or organ. Cell behaviour is almost always dependent on growth signals from the surrounding (mitogenic), which trigger cell division. These external growth factors (or ligands) bind to membrane-bound glycoprotein receptors that transmit the message via a series of intracellular signals that promote or inhibit the expression of specific genes. Examples of growth signals include diffusible growth factors, extracellular matrix proteins and cell-cell adhesion/interaction molecules. If these growth signals are absent, any typical normal cell will change to a quiescent state instead of active division. This dependence on exogenous growth factors is a critical homeostatic mechanism to control cell behaviour within a tissue. Cancer cells, on the other hand, generate mutant proteins (oncogenic proteins) which mimic these normal growth signals (proto-oncogenic proteins). Transformation of proto-oncogenes into oncogenes is brought about by several factors such as mutations, chromosomal rearrangements, viral insertion, gene amplifications etc. The consequence of oncogenic transformation is that tumour cells become independent of these external growth signaling factors in any normal tissue microenvironment. This acquired feature by tumour cells can be demonstrated empirically in vitro. Normal cells (e.g. skin fibroblast cells) that have been cultured in a petridish in vitro, will not divide and proliferate in the absence of growth factors found in serum. Tumour cells on the other hand can actively proliferate without depending on these growth factors. This autonomy from growth factor signaling leads to unregulated growth (such as in the absence of ideal conditions for cell division or stress) and increases the chances of acquiring further mutations in the cell genome. There are three main cellular strategies used by cancer cells in achieving growth factor autonomy, based on the growth factor signaling pathway

- Changes in extracellular growth signals
- Changes in transcellular mediators of those signals (receptors)
- Changes in intracellular signaling messengers that stimulate proliferation.

Changes in extracellular growth signals : Most soluble mitogenic growth factors (GFs) are heterotypic (made by one cell type in order to stimulate proliferation of another), but many cancer cells show autocrine stimulation (generate GF for its own cell type), thereby reducing dependence on GFs from other cells within the tissue. Many growth factor receptors, cytoplasmic and nuclear downstream effectors have been identified as oncogenes. The production of PDGF (platelet derived growth factor) by a type of brain tumour called glioblastoma, is one example of this. Cells infected with a viral oncoprotein v-sis tend to release large amounts of PDGF-like oncoproteins (PDGF- β , which attach to PDGF receptors of the same cell. This causes an autocrine signaling loop in which the cell generates its own growth factor signal resulting in constitutive growth stimulation.

Changes in transcellular signals (receptors) : The cell surface receptor that transduces growth stimulatory signals into the cell interior are themselves targets of deregulation during tumor pathogenesis. Mutations or changes in the number or types of receptors expressed by a cell can transform it into a cancer cell. The most common group of receptors implicated in several cancers belongs to the tyrosine kinase family. These include the EGF, FGF, IGF, PDGF receptors (Epidermal Growth Factor, Fibroblast Growth Factor, Insulin Growth Factor and Platelet Derived Growth Factor respectively). Some examples of these changes can include

- **Over expression :** GF receptors are often over expressed in many cancers, resulting in the cancer cells becoming hyper-responsive to levels of GF that would not normally trigger cell division. For example, in certain types of breast cancer (mammary carcinomas), the HER2/neu receptor is over expressed resulting in hyper proliferation.
- **Ligand-independent signaling :** GF-independent signaling can be brought about either by gross over expression of receptors or by structural changes in the receptor. For example, a truncated Epidermal Growth Factor Receptor (due to deletion in exons 2-7 of the extracellular domain) still sends growth-stimulating signals inside the cells consistently and without any EGF binding.
- **Receptor switching :** Cancer cells can also switch the types of receptors they express. For example – Cancer cells preferentially express extracellular matrix receptors (integrins) that favour growth. Integrins are bifunctional cell surface receptors that link cells to extracellular superstructures known as the extracellular matrix (ECM). Signaling molecules in the ECM enables the integrin receptors to transmit signals into the cytoplasm that influence cell behaviour, ranging from quiescence in normal tissue to motility, resistance to cell death, and entry into the active cell cycle in cancer cells.

Changes in intracellular signaling pathways that stimulate proliferation : The most common but complex mechanism of cancer transformation derives from changes in components of the intracellular, cytoplasmic signaling cascade, which receives input from the ligand-activated growth factor receptors on the cell membrane. Mutations in proteins belonging to the intracellular signaling cascade (adaptor proteins) are found in a majority of all human tumours. Examples of mutations in each component of this intracellular signaling

pathway in human cancers are highlighted below.

- **Cell cycle related kinase (c-crk)** : These small adaptor proteins (~60 amino acid residues) were first identified as a conserved sequence in the non-catalytic part of several cytoplasmic tyrosine kinases such as Abl and Src and have subsequently also been identified in several other protein families such as phospholipase, PI3-kinase, ras GTPase activating protein, adaptor proteins, CDC24 and CDC25. These proteins contain only SH3 and SH2 domains, of which the SH2 domains bind phosphorylated tyrosine residues while the SH3 domains bind proline-rich sequences in ligand proteins. The main function of the C-crk proteins is to bring substrate proteins to the tyrosine kinase receptors (TKR). The viral oncoprotein, Bcr-Abl, acts as a substrate for c-crk, causing sustained activation of the tyrosine kinase receptors and resulting in cell proliferation. Activation of TKR by the c-crk proteins has been implicated in some human chronic myelogenous leukemias (CML).
- **Ras (rat sarcoma)** : Ras is arguably one of the most well studied oncoprotein and is a component of the SOS-Ras-Raf-MAP kinase mitogenic cascade. About 25% of human tumors and 90% of all pancreatic tumours have a mutant form of the intracellular signaling molecule Ras (a small GTP binding signaling molecule). Ras proteins bind to GDP in an inactive state but upon stimulation through GEFs (guanine nucleotide exchange factors) are activated by binding to GTP. Active Ras functions as a GTPase through GAPs (GTPase-activating proteins), which hydrolyse the GTP and reach the inactive state. In tumours, typically, a single point mutation transforms the normal Ras into a Ras oncoprotein, in which the GTPase activity is lost, leading to a constantly active Ras and a cancer cell that cannot turn off its proliferation status. Alternatively, Ras activation pathways may be disrupted either due to oncoproteins or mutations in regulatory pathways.

For the past two decades, there have been intense studies on the signaling pathways involved in cell growth and proliferation. New targets upstream and downstream of key pathways are being discovered, revised and interlinked with each other, enabling extracellular signals to elicit multiple biological effects. One way of looking at the complexity of signaling pathways is by thinking of an electronic circuit board, where transistors are replaced by key regulatory proteins which act as binary switches (such as phosphates and kinases). Signals from either outside or inside the cell transmit messages of growth or quiescence, survival or death.

Tumor suppressor genes : Similar to GF signaling pathways, signals that normally suppress/block cell division are also received by cell-surface receptors that are coupled to intracellular signaling pathways. The genes that encode this class of proteins involved in restraining normal cell division are termed tumour suppressor genes. These anti-growth signals are closely linked to the cell cycle clock, which controls progressive cell division through mitosis. These are normal genes that slow down cell division, repair DNA errors,

and tell cells when to die (a process known as apoptosis or programmed cell death). Tumor suppressor genes code for anti-proliferation signals and proteins that suppress mitosis and cell growth. Signals from the external or internal environment dictate whether the cell should divide, or undergo quiescence, or enter the post-mitotic state or destroy itself. For example, presence of proteins involved in DNA replication at the end of the G1 phase pushes the cell into the S phase, whereas severe DNA damage can trigger the cell to kill itself by apoptosis or undergo quiescence. These checks provide a critical homeostatic mechanism for the cell to progress to cell division at the right time and under optimal growth conditions. Generally, tumor suppressors are transcription factors that are activated by cellular stress or DNA damage. Often DNA damage will cause the presence of free-floating genetic material as well as other signs, and will trigger enzymes and pathways which lead to the activation of tumor suppressor genes. The functions of such genes is to arrest the progression of the cell cycle in order to carry out DNA repair, preventing mutations from being passed on to daughter cells.

Typically, anti-growth signals work in two distinct ways :

- Forcing actively dividing cells into the quiescent (G0) phase of the cell cycle, which can be a temporary measure until there is a change in proliferative capacity (either a change in microenvironment conditions or there is a GF signal).
- Cells may be induced into a permanent post-mitotic (non-dividing) state as a result of development. For example the specific terminal differentiation of neurons or the denucleation state of mature erythrocytes.

Cancer cells, on the other hand, bypass or evade these anti-growth signals to enable their own growth and proliferation. For example, mutations in genes that normally inhibit cell proliferation would result in increased cell division. These tumour suppressor genes (TSGs) constitute a large group of genes that encode proteins whose normal role is to restrain cell division. Mutations in these genes lead to a loss-of function and typically, both copies (alleles) of the gene need to be altered to enable tumour formation (unlike oncogenes, which are gain-of-function mutation). One well-studied example of a tumour suppressor protein is the retinoblastoma (Rb) protein, involved in the formation of rare paediatric tumours found in the retina of the eye. Most mutations in the Rb gene involve gross chromosomal changes in the 3kb coding region of the gene and about a third tend to be single base change mutations. At the molecular level, the Rb protein (pRb) and its two relatives, p107 and p130, arguably regulate most of the anti-growth signals in a cell. In a hypophosphorylated state, pRb blocks proliferation by sequestering and altering the function of a key transcription factor called E2F, which control the expression of a multitude of genes essential for cell cycle progression from G1 into S phase. Alteration of the pRb pathway (either due to mutations or hyperphosphorylation of pRb) releases E2F, resulting in expression of genes involved in cell proliferation. Cells can also become insensitive to antigrowth factors that normally operate along this pathway to regulate cell cycle progression. The pRb signaling circuit can be disrupted in a variety of ways in different types of human tumors. For example, down

regulation/disruption of receptors and signaling molecules upstream of the pRb circuitry or the loss of functional pRb through mutations. Alternatively, in certain DNA virus-induced tumors, such as cervical carcinomas, viral oncoproteins, such as the E7 oncoprotein of human papilloma virus sequester pRb resulting in loss of function.

The p53 protein (so called because it is a tumour specific nuclear antigen of 53 kD size) another well-known example of a tumour suppressor protein. Originally discovered by David Lane, Arnold Levine and William Old in 1979, it has been termed 'guardian of the genome' because of its singularly critical role in the cell cycle. Almost 50% of all cancers show mutations in p53 and nearly 80% of mutations in Squamous Cell Carcinoma (SCC) involve p53. Mutations or loss of function of this vital protein leads to continued cell division despite the cell having damaged DNA (through radiation or mutagens for example). The role of p53 as a tumour suppressor was determined by two observations

- Mice which have both copies (alleles) of the p53 gene knocked out (p53^{-/-} mice) are prone to developing tumours (although interestingly, these mice are also prone to rapid ageing).
- Transfection (insertion) of wild type p53 genes into tumours of p53^{-/-} mice stopped tumour growth dramatically, thereby confirming the function of p53 as a tumour suppressor gene.

p53 is usually degraded in a normal cell. However, conditions of cellular stress or DNA damage (for example exposure to radiation or mutagens) result in expression of intermediate proteins which stabilize the p53 protein. This stable p53 protein forms a tetramer (a complex of 4 p53 proteins), which acts as a transcription factor and expresses genes involved in either halting cell cycle progression, or DNA repair (if the damage is minor) or inducing cell death (if the damage is too severe). In tumours however, the loss of function of p53 removes this essential 'guardianship', allowing the cell to continue dividing despite the DNA damaging mutations. Despite nearly half of all cancers possibly involving alterations in p53, its tumor suppressor function is poorly understood. p53 clearly has two functions: one a nuclear role as a transcription factor, and the other a cytoplasmic role in regulating the cell cycle, cell division, and apoptosis. The Warburg hypothesis is the preferential use of glycolysis for energy to sustain cancer growth. p53 has been shown to regulate the shift from the respiratory to the glycolytic pathway. However, a mutation can damage the tumor suppressor gene itself, or the signal pathway which activates it, "switching it off". The invariable consequence of this is that DNA repair is hindered or inhibited: DNA damage accumulates without repair, inevitably leading to cancer. Mutations of tumor suppressor genes that occur in germline cells are passed along to offspring, and increase the likelihood for cancer diagnoses in subsequent generations. Members of these families have increased incidence and decreased latency of multiple tumors. The tumor types are typical for each type of tumor suppressor gene mutation, with some mutations causing particular cancers, and other mutations causing others. The mode of inheritance of mutant tumor suppressors is that an affected member inherits a defective

copy from one parent, and a normal copy from the other. For instance, individuals who inherit one mutant p53 allele (and are therefore heterozygous for mutated p53) can develop melanomas and pancreatic cancer, known as Li-Fraumeni syndrome. Other inherited tumor suppressor gene syndromes include Rb mutations, linked to retinoblastoma, and APC gene mutations, linked to adenomatous polyposis coli (APC) colon cancer. Adenomatous polyposis coli is associated with thousands of polyps in colon while young, leading to colon cancer at a relatively early age. Finally, inherited mutations in BRCA1 and BRCA2 lead to early onset of breast cancer. Development of cancer was proposed in 1971 to depend on at least two mutational events. In what became known as the Knudson two-hit hypothesis, an inherited, germ-line mutation in a tumor suppressor gene would only cause cancer if another mutation event occurred later in the organism's life, inactivating the other allele of that tumor suppressor gene. Usually, oncogenes are dominant, as they contain gain-of-function mutations, while mutated tumor suppressors are recessive, as they contain loss-of-function mutations. Each cell has two copies of the same gene, one from each parent, and under most cases gain of function mutations in just one copy of a particular proto-oncogene is enough to make that gene a true oncogene. On the other hand, loss of function mutations needs to happen in both copies of a tumor suppressor gene to render that gene completely non-functional. However, cases exist in which one mutated copy of a tumor suppressor gene can render the other, wild-type copy are committed to non-functional. This phenomenon is called the dominant negative effect and is observed in many p53 mutations. Knudson's two hit model has recently been challenged by several investigators. Inactivation of one allele of some tumor suppressor genes is sufficient to cause tumors. This phenomenon is called haplo insufficiency and has been demonstrated by a number of experimental approaches. Tumors caused by haplo insufficiency usually have a later age of onset when compared with those by a two hit process. When tumor suppressor genes don't work properly, cells can grow out of control, which can lead to cancer. It may be helpful to think of a cell as a car. For it to work properly, there need to be ways to control how fast it goes. A proto-oncogene normally functions in a way that is similar to a gas pedal – it helps the cell grow and divide. An oncogene could be compared to a gas pedal that is stuck down, which causes the cell to divide out of control. A tumor suppressor gene is like the brake pedal on a car. It normally keeps the cell from dividing too quickly just as a brake keeps a car from going too fast. When something goes wrong with the gene, for example if a mutation causes it to stop working, cell division can get out of control. Slowly, medical scientists are identifying the oncogenes and tumor suppressor genes that are damaged by chemicals or radiation and those that, when inherited, can lead to cancer. For example, the 1990s discovery of 2 genes that cause some breast cancers, BRCA1 and BRCA2, is a step forward because these genes can be used to identify people who have a higher risk of developing breast cancer.

Another strategy used by cancer cells is to avoid the irreversible terminal differentiation of cells into postmitotic states. One example of this method involves the transcription factor c-Myc, which stimulates growth during normal development by associating with another factor, Max. To induce differentiation however, Max forms complexes with Mad (Mad-Max

complexes) to trigger differentiation-inducing signals. However, in certain tumours, the c-Myc oncoprotein is over expressed, thereby shifting the balance to favour Myc–Max complexes, thereby inhibiting differentiation and promoting growth. Although the interlinking between the various growth and differentiation-inducing signals with the cell division cycle are still being clarified, it is clear that understanding the antigrowth signaling circuitry is vital to understanding cancer development.

Other genes have been discovered that are linked to cancers that run in families, such as cancers of the colon, rectum, kidney, ovary, thyroid, pancreas, and skin melanoma. Familial cancer is not nearly as common as spontaneous cancer (cancer that is caused by DNA damage that starts during a person's lifetime). Cancer linked to heredity is less than 15% of all cancers. Still, it's important to understand these cancers because with continued research in genetics we may be able to identify more people at very high risk. Once researchers recognized the importance of specific genetic changes in cancer, they soon began working to develop targeted therapies (drugs or substances that interfere with specific molecules) to overcome the effects of these changes in tumor suppressor genes and oncogenes.

History of cancer epidemiology :

During the 18th century, 3 important observations launched the field of cancer epidemiology

- In 1713, Bernardino Ramazzini, an Italian doctor, reported the virtual absence of cervical cancer and relatively high incidence of breast cancer in nuns and wondered if this was in some way related to their celibate lifestyle. This observation was an important step toward identifying and understanding the importance of hormones (like the changes that come with pregnancy) and sexually-transmitted infections and cancer risk.
- In 1775, Percival Pott of Saint Bartholomew's Hospital in London described an occupational cancer in chimney sweeps, cancer of the scrotum, which was caused by soot collecting in the skin folds of the scrotum. This research led to many more studies that identified a number of occupational carcinogenic exposures and led to public health measures to reduce a person's cancer risk at work.
- Thomas Venner of London was one of the first to warn about tobacco dangers in his *Via Recta*, published in London in 1620. He wrote that "immoderate use of tobacco hurts the brain and the eye and induces trembling of the limbs and the heart." And 150 years later, in 1761, only a few decades after recreational tobacco became popular in London, John Hill wrote a book entitled "Cautions against the Immoderate Use of Snuff". These first observations linking tobacco and cancer led to epidemiologic research many years later (in the 1950s and early 1960s) which showed that smoking causes lung cancer and led to the US Surgeon General's 1964 report *Smoking and Health*.

Epidemiologists continue to search for factors that cause cancer (like tobacco use, obesity, ultraviolet radiation), as well as those things that can help protect against cancer

(such as physical activity and a healthy diet). This research provides evidence to guide public health recommendations and regulations.

Genetic basis of cancer:

Due to connection between cancer and cell signaling, it is not surprising in hindsight, that many of the proximate causes of cancer are thought to be mutations in genes for signal transduction components. The first indications of this came from studies of DNA tumor viruses that infected non-human vertebrates. It was established that particular genes in the genomes of these viruses (termed oncogenes) were responsible for tumor promotion. With time and the convergence of cellular and viral oncology, it was realized that oncogenes were the mutant counterparts of normal cellular genes. The cellular genes are called proto-oncogenes. Many of the cellular counterparts of oncogenes have now been identified (and cloned), and it is clear that most are components of some form of signal transduction pathway. Many proto-oncogenes encode things like growth factors (eg. sis), growth factor receptors (eg. erb B), protein kinases (eg. src) and transcription factors (eg. jun). The signaling systems are used by cells to make decisions like grow/don't grow, move/don't move or change patterns of gene expression. Many of these cellular components are the same kinds of factors as well as driving forces behind the fundamental mechanisms of developmental decision making. Little wonder then that the mechanisms that control development and go out of control in cancer seem so intimately related. Characteristically, oncogenes derange growth regulation by hyper activating growth stimulatory signals; thus normal growth control is overridden. The mutations generally produce an over stimulation of a positive signal, only one mutant copy of the gene is required to provide the phenotype. Consistent with this, most oncogenes are dominant mutations.

There is, however, another way to get over stimulation of a positive signal. That is to have a recessive, lose of function, mutation in a negative regulator of the signal. This is the basis for another group of genes found to be mutant in many cancers; called tumor suppressor genes or anti-oncogenes (neither name is completely satisfying). One of the clearest indications of the existence of such genes came from experiments in which tumor-producing and non-tumor-producing cells were fused together. With rare exceptions (which turned out to be important) the fused cells were non-tumorigenic. That meant that something in the normal cells was blocking the ability of the tumor cells to grow (i.e. was suppressing tumor formation). When investigators looked at those unusual cases where the fused cells could produce tumors, they found that one or more chromosomes from the non-tumorigenic partner had been lost. This gave them the opportunity to map potential tumor suppressor genes to specific chromosomal locations and eventually clone a handful. Probably the most well understood (and the simplest) example of a tumor suppressor gene is the Rb gene associated with retinoblastoma. Based on chromosome lose in a number of independent and familial cases of retinoblastoma, it was predicted that a gene on chromosome 13 (13q14) was critical for the disease. In 1987, Lee and colleagues isolated a candidate gene that encoded a 4.7kb mRNA and a 105kD

protein. Subsequently this gene has been established as that missing in retinoblastoma patients. The protein encoded by the Rb locus (p105) is a nuclear protein that is found both in a phosphorylated and unphosphorylated state. The phosphorylation state of the protein is cell cycle dependent; maximal close to the start of S phase and lowest at the end of mitosis/beginning of G1. It is believed that a cell cycle “start” kinase phosphorylates p105. The phosphorylated protein then releases from a multi protein complex which can then initiate the cell cycle. When mitosis is complete a phosphatase removes the phosphate from p105, it reassociates with the complex and blocks further rounds of cell division. When the Rb gene is defective a functional p105 is not made and the protein complex that initiates the cell cycle never gets turned off, thus unregulated growth. Undoubtedly this is not the whole story, at least in part because p105 is present in all (or most) of the cells of the body but its loss only permits unregulated growth in specific cell types (retina). Clearly other players are involved with the signal normally transmitted through p105 but their identities are yet to be determined. A number of other tumor suppressor genes have recently been identified (eg. p53, NF-1 from neurofibromatosis, and WT-1 from Wilm’s tumors). The mechanisms by which they work are even less well understood than that of Rb. The study of tumor suppressor genes represents an area of enormous effort at the moment, and understanding their roles in regulating cell growth will likely provide significant insights into both cancer biology and embryogenesis. In the end it seems very likely that many of the kinds of integrated signals that are necessary for proper differentiation and pattern formation in the embryo will be important in understanding the control of cancer. By understanding developmental signaling it may be possible to derive noncytotoxic cures for carcinomas. Likewise a greater knowledge of embryonic development may derive from the use of cancer cells and understanding the nature of their malfunctions.

Process of cancer spreads in the body :

One of the things that make cancer so difficult to treat is the fact that it can spread around the body. Cancer cells break away from the original primary tumour and spread through the bloodstream or the lymphatic system, forming new secondary tumours in organs such as the lungs, liver or brain. Scientists call this process metastasis. Most of the cells in our bodies stick to their neighbours through the help of ‘Velcro-like’ molecules on their surface known as integrins. Integrins are vital for forming structured tissues and organs, like the skin and the lungs. But they also help cancer cells that have broken away from a tumour to take root elsewhere in the body. Professor Ian Hart at Barts and The London School of Medicine and Dentistry has discovered that a particular integrin is found in some aggressive cancers that have a poor outlook. His work could lead to ways to detect cancer once it has spread and, one day, even help to prevent the process in the first place. In Edinburgh, Professor Maggie Frame is using cutting-edge imaging techniques to watch the effect of drugs that block two key proteins involved in cancer spread, to see if the drugs can keep cancer cells at bay. In order to spread, some cells from the primary cancer must break away, travel to another part of the body and start growing there. Cancer cells do not stick together as well as normal cells do.

They may also produce substances that stimulate them to move. There are three main ways a cancer spreads:

- Local spread
- Through the blood circulation
- Through the lymphatic system

Local spread : The cancer spreads from where it began by growing into nearby areas. As a tumour gets bigger, it takes up more and more room in the body. Soon it begins to grow into the body structures nearby. This is called local invasion. How a cancer actually grows into surrounding normal body tissues is not fully understood. But research has pointed to three ways that the tumour is most likely to do this

- Pressure from the growing tumour
- Using enzymes
- Cancer cells moving through the tissue

A particular tumour will probably use all three of these ways of spreading. Which way is used most will depend partly on the type of tumour and partly on where in the body it is growing.

Pressure from the growing tumour : As the tumour grows and takes up more space, it begins to press on the normal body tissue nearby. The finger like appearance of the growth happens because it is easier for the growing cancer to force its way through some paths than others - for example, cancers may grow between sheets of muscle tissue rather than straight through one particular muscle. As the cancer grows, it will squeeze and block small blood vessels in the area. Due to low blood and oxygen levels, some of the normal tissue will begin to die off. This makes it easier for the cancer to continue to push its way through.

Using enzymes : Many normal blood cells produce chemicals called enzymes that break down cells and tissues. The blood cells use their enzymes to attack invading bacteria and viruses. They also use them to break down and clear up damaged areas in the body. The damaged cells have to be cleared away so that the body can replace them with new ones. This is all part of the natural healing process. Many cancers contain larger amounts of these enzymes than normal tissues. Some cancers also contain a lot of normal white blood cells. They are part of the body's immune response to the cancer. As the cancer pushes through and breaks down normal tissues, it may cause bleeding as it causes damage to nearby blood vessels.

Cancer cells moving through the tissue : One of the things that make cancer cells different to normal cells is that they can move about more easily. So it seems likely that one of the ways that cancers spread through nearby tissues is by the cells directly moving. Scientists have discovered a substance made by cancer cells which stimulates them to move. They don't know for sure yet, but it seems likely that this substance plays a big part in the local spread of cancers. This research is exciting because, if a substance has been found that helps cancer cells

move, then researchers can start to find ways to stop the substance working. They may also be able to find ways to stop the cancer cells making the substance in the first place. If cancers can be stopped from spreading, then it might be much easier to cure them.

A cancer probably just grows out in a random direction from the place where it started. However, tumours can spread into some tissues more easily than others. For example, large blood vessels that have very strong walls and dense tissues such as cartilage are hard for tumours to grow into. So locally, tumours grow along the ‘path of least resistance’. This means that they probably just take the easiest route.

Through the blood circulation:

The cancer spreads from where it began by getting into the blood. The cancer travels through the blood vessels to other parts of the body. In order to spread, the cancer cell must first become detached from the primary cancer. It must then move through the wall of a blood vessel to get into the bloodstream.

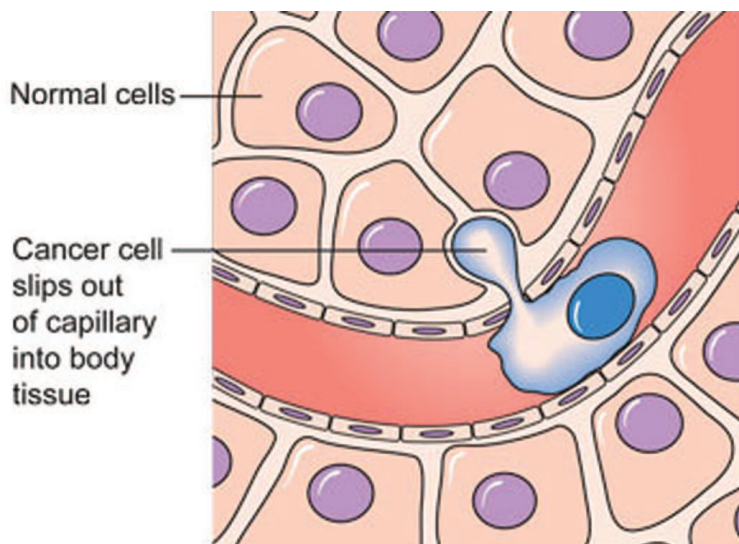


Fig: Cancer spread through the blood system

When it is in the bloodstream, it is swept along by the circulating blood until it gets stuck somewhere, usually in a very small blood vessel called a capillary. Then it must move back through the wall of the capillary and into the tissue of the organ close by. There it must start to multiply to grow a new tumour. Most cancer cells do not survive it. Probably, out of many thousands of cancer cells that reach the blood circulation only one will survive to form a secondary cancer or metastasis. Some cancer cells are probably killed off by the white blood cells in our immune system. Others cancer cells may die because they are battered around by the fast flowing blood. Cancer cells in the circulation may try to stick to platelets to form clumps to give themselves some protection. This may also help them to be filtered out in the

next capillary network they come across so they can then move into the tissues to start a secondary tumour.

Through the lymph system: The cancer spreads from where it began by getting into the lymph system. The cancer travels through the lymph vessels to other parts of the body. Cancer that starts in the lymph nodes is called lymphoma. Cancer can spread from where it started (the primary site) to other parts of the body. When cancer cells break away from a tumor, they can travel to other areas of the body through either the bloodstream or the lymph system. Cancer cells can travel through the bloodstream to reach distant organs. If they travel through the lymph system, the cancer cells may end up in lymph nodes. Either way, most of the escaped cancer cells die or are killed before they can start growing somewhere else. But one or two might settle in a new area, begin to grow, and form new tumors. This spread of cancer to a new part of the body is called metastasis. In order for cancer cells to spread to new parts of the body, they have to go through several changes. They first have to become able to break away from the original tumor and then attach to the outside wall of a lymph vessel or blood vessel. Then they must move through the vessel wall to flow with the blood or lymph to a new organ or lymph node. When cancer grows inside lymph nodes, it usually affects the lymph nodes near the tumor itself. These nodes are the ones that have been doing most of the work to filter out or kill the cancer cells.

Normal lymph nodes are tiny and can be hard to find, but when there's infection, inflammation, or cancer, the nodes can get larger. Those near the body's surface often get big enough to feel with your fingers, and some can even be seen. But if there are only a few cancer cells in a lymph node, it may look and feel normal. In that case, the doctor must check for cancer by removing all or part of the lymph node. When a surgeon operates to remove a primary cancer, one or more of the nearby (regional) lymph nodes may be removed as well. Removal of one lymph node is called a biopsy. When many lymph nodes are removed, it's called lymph node sampling or dissection. When cancer has spread to lymph nodes, there is a higher risk that the cancer might come back after surgery. This information helps the doctor decide whether more treatment, like chemo or radiation, might be needed after surgery. Doctors may also take samples of one or more nodes using needles. Usually, this is done on lymph nodes that are enlarged. This is called a needle biopsy. The tissue that's removed is looked at under the microscope by a pathologist (a doctor who diagnoses illness using tissue samples) to find out if there are cancer cells in it. Under the microscope, any cancer cells in the nodes look like cells from the primary tumor. For instance, when breast cancer spreads to the lymph nodes, the cells in the nodes look like breast cancer cells. The pathologist prepares a report, which details what was found. If a node has cancer in it, the report describes what it looks like and how much was seen. Doctors may also use scans or other imaging tests to look for enlarged nodes around a cancer if the nodes are deep in the body. If there's a lot of cancer in a node, the large mass can be seen easily. If the cancer is growing out of the lymph node through the layer of connective tissue on the outside (called the capsule), it's called

extracapsular extension. More cancer in the nodes may mean that the cancer is fast growing and/or more likely to spread to other places in the body. But if nearby lymph nodes are the only other place cancer is found beyond the main (primary) site, surgery to remove the main tumor and the nearby lymph nodes may be able to get rid of it all. Cancer that has spread to nodes further away from the primary cancer will more likely need extra treatment with chemo or radiation. For instance, if nodes are affected on the other side of the body, the cancer may need more treatment.

Why cancers spread where they do :

Whether it is in the blood or the lymph, the moving cancer cell stops at the first place it gets stuck. In the bloodstream, this is often the first capillary network it comes across. The blood flow from most body organs goes from the organ to the capillaries in the lungs. So not surprisingly, the lungs are a very common place for cancer to spread to. The blood from the organs of the digestive system goes through the capillaries of the liver before going back to the heart and then to the lungs. So it is common for digestive system cancers to spread to the liver. In fact, the liver is the second most common area of cancer spread.

Some cancers show unexpected patterns of spread. For example, prostate cancer often spreads to the bones. Scientists are still investigating why this happens. Cancer cells often get trapped in the group of lymph nodes closest to the tumour. During cancer surgery, the surgeon may remove the main lymph nodes close to the area of the cancer. For example, a surgeon operating to remove a breast cancer will remove one or more of the lymph nodes from under the arm. These are the first lymph nodes that the lymph draining from the breast goes to and so are most likely to contain any escaping cancer cells. The first nodes that lymph draining from a tumour reaches are called the sentinel lymph nodes. After removing sentinel nodes, the surgeon will send them off to the lab to see if they contain cancer cells.

Micrometastases : Micrometastases are areas of cancer spread (metastases) that are too small to see. If there are individual cells, or even small areas of growing cells elsewhere in the body, no scan is detailed enough to show them. For a few types of cancer, blood tests can detect certain proteins released by the cancer cells. These may give a sign that there are metastases too small to show up on a scan. But for most cancers, there is no blood test that can say whether a cancer has spread or not. For most cancers the doctor can only say whether it is likely or not that a patient has micrometastases. This 'best guess' may be based on the following factors.

- Previous experience of many other patients treated in the same way. Doctors naturally collect and publish this information to help each other
- Whether cancer cells are found in the blood vessels in the tumour removed during surgery (for example in testicular cancer). If they are found then cancer cells are more likely to have reached the bloodstream and spread to somewhere else in the body
- The grade of the cancer - the higher the grade, the more aggressive the cancer and the

more likely that cells have spread

- Whether lymph nodes removed during an operation contained cancer cells (for example in breast cancer or bowel cancer). If the lymph nodes contained cancer cells this shows that cancer cells have broken away from the original cancer. But there is no way of knowing whether they have spread to any other areas of the body.

This information is important. If the doctor thinks it is likely that there are micrometastases, they may offer further treatment such as chemotherapy, radiotherapy, biological therapy or hormone therapy. Treatment after surgery is called adjuvant treatment. The aim is to kill the areas of cancer cells before they grow big enough to be seen on a scan. Some doctors call this 'belt and braces' treatment. In other words, the treatment is to try to make sure the cancer does not come back. But no one can know for sure if all the cancer cells have been destroyed when someone has finished treatment. It is this uncertainty that can make cancer difficult to cope with for many people, even if they seem to have been successfully treated.

Treatment of cancer is based on the type of cancer a person has, and the stage of the cancer. Doctors use a system to assign a stage to the cancer. The most common staging system is the TNM system. The T in TNM stands for tumor, the M stands for metastasis, and the N stands for lymph nodes. If there's no cancer found in the lymph nodes near the cancer, the N is assigned a value of 0. If nearby or distant nodes show cancer, the N is assigned a number such as 1, 2 or sometimes 3, depending on how many nodes are affected, how much cancer is in them, how large they are, and where they are. A cancer with lower TNM numbers is usually easier to treat and has a better outlook for survival. For instance, a cancer with T1, N0, M0, would be a cancer that was found very early, before it spread. The T1 would mean a small tumor, the N0 means that no nodes are involved, and the M0 means that no metastases were found. Nodes that have been removed during cancer surgery can leave part of the body without a way to drain off the lymph fluid in the affected area. Many of the lymph vessels now run into a dead end where the node used to be, and fluid can back up. This is called lymphedema, and it can become a life-long problem. The more lymph nodes that are removed, the more likely it is to occur.

Cancer in the twenty-first century :

The growth in our knowledge of cancer biology has led to remarkable progress in cancer prevention, early detection, and treatment. Scientists have learned more about cancer in the last two decades than had been learned in all the centuries preceding. This doesn't change the fact, however, that all scientific knowledge is based on the knowledge already acquired by the hard work and discovery of our predecessors – and we know that there's still a lot more to learn. Cancer research is advancing on so many fronts that it's hard to choose the ones to highlight, but here are a few examples:

More targeted therapies : As more is learned about the molecular biology of cancer, researchers will have more targets for their new drugs. Along with more monoclonal antibodies and small signaling pathway inhibitors, researchers are developing new classes

of molecules such as antisense oligodeoxynucleotides and small interfering RNA (siRNA). Research is being done to develop targeted drugs that are aimed at proteins produced by specific gene mutations in cancer cells, too.

Immunotherapy : Drugs aimed at specific immune checkpoints are being developed to help the immune system better kill cancer cells. More on cancer genetics: Researchers are looking for gene mutations that cause some patients to respond better to certain drugs.

Nanotechnology : New technology for producing materials that form extremely tiny particles is leading to very promising imaging tests that can more accurately show the location of tumors. It also is aiding the development of new ways to deliver drugs more specifically and effectively to cancer cells.

Robotic surgery : This term refers to manipulation of surgical instruments remotely by robot arms and other devices controlled by a surgeon. Robotic systems have been used for several types of cancer surgery; radical prostatectomy is among the most common uses in surgical oncology. As mechanical and computer technology improve, some researchers expect future systems will be able to remove tumors more completely and with less surgical trauma.

Expression profiling and proteomics : Expression profiling lets scientists determine relative output of hundreds or even thousands of molecules (including the proteins made by RNA, DNA, or even a cell or tissue) at one time. Knowing what proteins are present in cells can tell scientists a lot about how the cell is behaving. In cancer, it can help distinguish more aggressive cancers from less aggressive ones, and can often help predict which drugs the tumor is likely to respond to. Proteomic methods are also being tested for cancer screening. For most types of cancer, measuring the amount of one protein in the blood is not very good at finding early cancers. But researchers are hopeful that comparing the relative amounts of many proteins may be more useful, and that finding large amounts of certain proteins and less of others can provide accurate, useful information about cancer treatment and its outcomes. Proteins (and other types of molecules) are even found in exhaled breath, which is now being tested to find out if it can show early signs of lung cancer. This is an exciting area of research and early results in lung and colorectal cancer studies have been promising.

Different types of cancer :

Acute granulocytic leukemia (Leukemia)	Infiltrating lobular carcinoma (ILC) (Breast cancer)	Pancreatic cancer
Acute lymphocytic leukemia (ALL) (Leukemia)	Inflammatory breast cancer (IBC) (Breast cancer)	Papillary carcinoma (Breast cancer)
Acute myelogenous leukemia (AML) (Leukemia)	Intestinal Cancer	Paranasal sinus cancer
Adenocarcinoma (Lung cancer)	Intrahepatic bile duct cancer (Bile duct cancer)	Parathyroid cancer (Thyroid cancer)

Adenosarcoma (Lung cancer)	Invasive / infiltrating breast cancer (Breast cancer)	Pelvic cancer
Adrenal cancer	Islet cell cancer (Pancreatic cancer)	Penile cancer
Adrenocortical carcinoma (Adrenal cancer)	Jaw cancer (Oral cancer)	Peripheral nerve cancer (Brain cancer)
Anal cancer	Kaposi sarcoma (Soft tissue sarcoma)	Peritoneal cancer (Ovarian cancer)
Anaplastic astrocytoma (Brain cancer)	Kidney cancer	Pharyngeal cancer (Throat cancer)
Angiosarcoma (Soft tissue sarcoma)	Laryngeal cancer (Throat cancer)	Pheochromocytoma (Adrenal cancer)
Appendix cancer	Leiomyosarcoma (Soft tissue sarcoma)	Pilocytic astrocytoma (Brain cancer)
Astrocytoma (Brain cancer)	Leptomeningeal metastases	Pineal region tumor (Brain cancer)
Basal cell carcinoma (Skin cancer)	Leukemia	Pineoblastoma
B-Cell lymphoma (Non-Hodgkin lymphoma (NHL))	Lip cancer (Oral cancer)	Pituitary gland cancer (Brain cancer)
Bile duct cancer	Liposarcoma (Soft tissue sarcoma)	Primary central nervous system (CNS) lymphoma
Bladder cancer	Liver cancer	Prostate cancer
Bone cancer	Lobular carcinoma in situ (Breast cancer)	Rectal cancer (Colorectal cancer)
Bowel cancer (Colorectal cancer)	Low-grade astrocytoma (Brain cancer)	Renal cell cancer (Kidney cancer)
Brain cancer	Lung cancer	Renal pelvis cancer (Kidney cancer)
Brain stem glioma (Brain cancer)	Lymph node cancer (Non-Hodgkin lymphoma (NHL))	Rhabdomyosarcoma (Soft tissue sarcoma)
Brain tumor (Brain cancer)	Lymphoma (Non-Hodgkin lymphoma (NHL))	Salivary gland cancer (Oral cancer)
Breast cancer	Male breast cancer (Breast cancer)	Sarcoma (Soft tissue sarcoma)
Carcinoid tumors	Medullary carcinoma (Breast cancer)	Sarcoma, bone (Bone cancer)
Cervical cancer	Medulloblastoma (Brain cancer)	Sarcoma, soft tissue
Chondrosarcoma (Bone cancer)	Melanoma	Sarcoma, uterine (Uterine cancer)

Chronic lymphocytic leukemia (CLL) (Leukemia)	Meningioma (Brain cancer)	Sinus cancer
Chronic myelogenous leukemia (CML) (Leukemia)	Merkel cell carcinoma (Skin cancer)	Skin cancer
Colon cancer (Colorectal cancer)	Mesenchymal chondrosarcoma (Bone cancer)	Small cell lung cancer (SCLC) (Lung cancer)
Colorectal cancer	Mesenchymous	Small intestine cancer
Craniopharyngioma (Brain cancer)	Mesothelioma	Soft tissue sarcoma
Cutaneous lymphoma (Skin cancer)	Metastatic breast cancer (Breast cancer)	Spinal cancer
Cutaneous melanoma (Melanoma)	Metastatic melanoma (Melanoma)	Spinal column cancer (Spinal cancer)
Diffuse astrocytoma (Brain cancer)	Metastatic squamous neck cancer	Spinal cord cancer (Spinal cancer)
Ductal carcinoma in situ (DCIS) (Breast cancer)	Mixed gliomas (Brain cancer)	Spinal tumor (Spinal cancer)
Endometrial cancer (Uterine cancer)	Mouth cancer (Oral cancer)	Squamous cell carcinoma (Skin cancer)
Ependymoma (see Brain cancer)	Mucinous carcinoma (Breast cancer)	Stomach cancer
Epithelioid sarcoma (Soft tissue sarcoma)	Mucosal melanoma (Oral cancer)	Synovial sarcoma (Soft tissue sarcoma)
Esophageal cancer	Multiple myeloma	T-cell lymphoma (Non-Hodgkin lymphoma (NHL))
Ewing sarcoma (Bone cancer)	Nasal cavity cancer (Throat cancer)	Testicular cancer
Extrahepatic bile duct cancer (Bile duct cancer)	Nasopharyngeal cancer (Throat cancer)	Throat cancer
Eye cancer	Neck cancer (Head and neck cancer)	Thymoma/thymic carcinoma
Fallopian tube (Ovarian cancer)	Neuroblastoma	Thyroid cancer
Fibrosarcoma (Soft tissue sarcoma)	Neuroendocrine tumors (Intestinal Cancer)	Tongue cancer (Oral cancer)
Gallbladder cancer	Non-Hodgkin lymphoma (NHL)	Tonsil cancer
Gastric cancer (see Stomach cancer)	Non-Hodgkin's lymphoma (Non-Hodgkin lymphoma)	Transitional cell cancer (Bladder cancer)
Gastrointestinal cancer	Non-small cell lung cancer (NSCLC) (Lung cancer)	Transitional cell cancer (Kidney cancer)

Gastrointestinal carcinoid cancer	Oat cell cancer (Lung cancer)	Transitional cell cancer (Ovarian cancer)
Gastrointestinal stromal tumors (GIST)	Ocular cancer	Triple-negative breast cancer (Breast cancer)
General	Ocular melanoma	Tubal cancer
Germ cell tumor (Brain cancer)	Oligodendroglioma (Brain cancer)	Tubular carcinoma (Breast)
Glioblastoma multiforme (GBM) (Brain cancer)	Oral cancer	Ureteral cancer (Bladder cancer)
Glioma (Brain cancer)	Oral cavity cancer (Oral cancer)	Ureteral cancer (Kidney cancer)
Hairy cell leukemia (Leukemia)	Oropharyngeal cancer (Throat)	Urethral cancer
Head and neck cancer	Osteogenic sarcoma (Bone cancer)	Uterine adenocarcinoma (Uterine cancer)
Hemangioendothelioma	Osteosarcoma (Bone cancer)	Uterine cancer
Hodgkin lymphoma	Ovarian cancer	Uterine sarcoma (Uterine cancer)
Hodgkin's disease (Hodgkin lymphoma)	Ovarian epithelial cancer (Ovarian cancer)	Vaginal cancer
Hodgkin's lymphoma (Hodgkin lymphoma)	Ovarian germ cell tumor (Ovarian cancer)	Vulvar cancer
Hypopharyngeal cancer (Throat cancer)	Ovarian primary peritoneal carcinoma (Ovarian cancer)	
Infiltrating ductal carcinoma (IDC) (Breast cancer)	Paget's disease (Breast cancer)	

Breast cancer :

Breast cancer is a malignant tumor that starts in the cells of the breast. Breast cancer usually starts off in the inner lining of milk ducts or the lobules that supply them with milk. A breast cancer that started off in the lobules is known as lobular carcinoma, while one that developed from the ducts is called ductal carcinoma. A malignant tumor is a group of cancer cells that can grow into (invade) surrounding tissues or spread (metastasize) to distant areas of the body. The disease occurs almost entirely in women, but men can get it, too. The female breast is made up mainly of lobules (milk-producing glands), ducts (tiny tubes that carry the milk from the lobules to the nipple), and stroma (fatty tissue and connective tissue surrounding the ducts and lobules, blood vessels, and lymphatic vessels). Most breast cancers begin in the cells that line the ducts (ductal cancers). Some begin in the cells that line the lobules (lobular cancers), while a small number start in other tissues. A mature human female's breast consists of fat, connective tissue and thousands of lobules - tiny glands which produce milk. The milk of a breastfeeding mother goes through tiny ducts (tubes) and is delivered through the nipple.

The breast, like any other part of the body, consists of billions of microscopic cells. These cells multiply in an orderly fashion - new cells are made to replace the ones that died. In cancer, the cells multiply uncontrollably, and there are too many cells, progressively more and more than there should be. Cancer that begins in the lactiferous duct (milk duct), known as ductal carcinoma, is the most common type. Cancer that begins in the lobules, known as lobular carcinoma, is much less common.

Breast cancer is the most common invasive cancer in females worldwide. It accounts for 16% of all female cancers and 22.9% of invasive cancers in women. 18.2% of all cancer deaths worldwide, including both males and females, are from breast cancer. Breast cancer rates are much higher in developed nations compared to developing ones. There are several reasons for this, with possibly life-expectancy being one of the key factors - breast cancer is more common in elderly women; women in the richest countries live much longer than those in the poorest nations. The different lifestyles and eating habits of females in rich and poor countries are also contributory factors, experts believe. According to the National Cancer Institute, 232,340 female breast cancers and 2,240 male breast cancers are reported in the USA each year, as well as about 39,620 deaths caused by the disease.

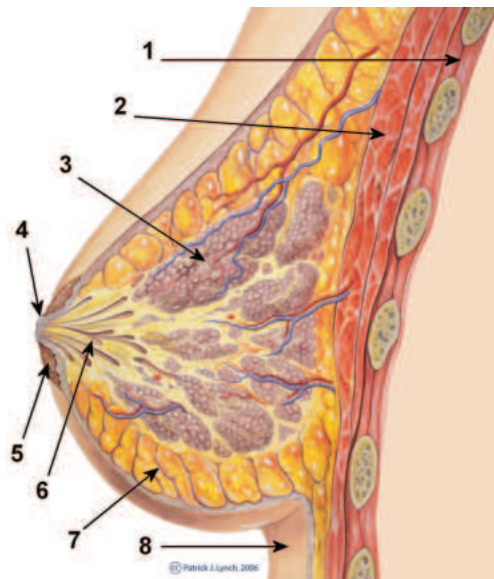


Fig: Anatomy of female breast. 1. Chest wall. 2. Pectoralis muscles. 3. Lobules (glands that make milk). 4. Nipple surface. 5. Areola. 6. Lactiferous duct tube that carries milk to the nipple. 7. Fatty tissue. 8. Skin.

In case of invasive breast cancer the cancer cells break out from inside the lobules or ducts and invade nearby tissue. With this type of cancer, the abnormal cells can reach the lymph nodes, and eventually make their way to other organs (metastasis), such as the bones, liver or lungs. The abnormal (cancer) cells can travel through the bloodstream or the lymphatic system to other parts of the body; either early on in the disease, or later. While in case of non-invasive breast cancer, the cancer is still inside its place of origin and has not

broken out. Lobular carcinoma in situ is when the cancer is still inside the lobules, while ductal carcinoma in situ is when they are still inside the milk ducts. “In situ” means “in its original place”. Sometimes, this type of breast cancer is called “pre-cancerous”; this means that although the abnormal cells have not spread outside their place of origin, they can eventually develop into invasive breast cancer.

The lymph (lymphatic) system of the breast

The lymph system is important to understand because it is one way breast cancers can spread. This system has several parts. Lymph nodes are small, bean-shaped collections of immune system cells (cells that are important in fighting infections) that are connected by lymphatic vessels. Lymphatic vessels are like small veins, except that they carry a clear fluid called lymph (instead of blood) away from the breast. Lymph contains tissue fluid and waste products, as well as immune system cells. Breast cancer cells can enter lymphatic vessels and begin to grow in lymph nodes. Most lymphatic vessels in the breast connect to lymph nodes under the arm (axillary nodes). Some lymphatic vessels connect to lymph nodes inside the chest (internal mammary nodes) and either above or below the collarbone (supraclavicular or infraclavicular nodes). If the cancer cells have spread to lymph nodes, there is a higher chance that the cells could have also gotten into the bloodstream and spread (metastasized) to other sites in the body. The more lymph nodes with breast cancer cells, the more likely it is that the cancer may be found in other organs as well. Because of this, finding cancer in one or more lymph nodes often affects the treatment plan. Still, not all women with cancer cells in their lymph nodes develop metastases, and some women can have no cancer cells in their lymph nodes and later develop metastases.

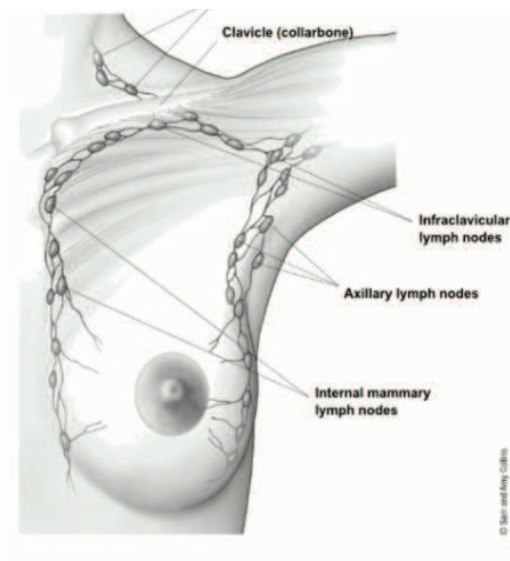


Fig: lymph nodes in relation to the breast

Benign breast lumps : Most breast lumps are not cancerous (benign). Still, some may

need to be biopsied (sampled and viewed under a microscope) to prove they are not cancer.

Fibrosis and cysts : Most lumps turn out to be caused by fibrosis and/or cysts, benign changes in the breast tissue that happen in many women at some time in their lives. (This is sometimes called fibrocystic changes and used to be called fibrocystic disease.) Fibrosis is the formation of scar-like (fibrous) tissue, and cysts are fluid-filled sacs. These conditions are most often diagnosed by a doctor based on symptoms, such as breast lumps, swelling, and tenderness or pain. These symptoms tend to be worse just before a woman's menstrual period is about to begin. Her breasts may feel lumpy and, sometimes, she may notice a clear or slightly cloudy nipple discharge.

Fibroadenomas and intraductal papillomas : Benign breast tumors such as fibroadenomas or intraductal papillomas are abnormal growths, but they are not cancerous and do not spread outside the breast to other organs. They are not life threatening. Still, some benign breast conditions are important because women with these conditions have a higher risk of developing breast cancer. Here are some of the key words used to describe breast cancer

Carcinoma : This is a term used to describe a cancer that begins in the lining layer (epithelial cells) of organs like the breast. Nearly all breast cancers are carcinomas (either ductal carcinomas or lobular carcinomas).

Adenocarcinoma : An adenocarcinoma is a type of carcinoma that starts in glandular tissue (tissue that makes and secretes a substance). The ducts and lobules of the breast are glandular tissues (they make breast milk), so cancers starting in these areas are often called adenocarcinomas.

Carcinoma in situ : This term is used for an early stage of cancer, when it is confined to the layer of cells where it began. In breast cancer, in situ means that the cancer cells remain confined to ducts (ductal carcinoma in situ). The cells have not grown into (invaded) deeper tissues in the breast or spread to other organs in the body. Ductal carcinoma in situ of the breast is sometimes referred to as non-invasive or pre-invasive breast cancer because it might develop into an invasive breast cancer if left untreated. When cancer cells are confined to the lobules it is called lobular carcinoma in situ.

Invasive (infiltrating) carcinoma : An invasive cancer is one that has already grown beyond the layer of cells where it started (as opposed to carcinoma in situ). Most breast cancers are invasive carcinomas—either invasive ductal carcinoma or invasive lobular carcinoma.

Sarcoma : Sarcomas are cancers that start in connective tissues such as muscle tissue, fat tissue, or blood vessels. Sarcomas of the breast are rare. The diagnosis of breast cancer is confirmed by taking a biopsy of the concerning lump. Once the diagnosis is made, further tests are done to determine if the cancer has spread beyond the breast and which treatments it may respond to. The balance of benefits versus harms of breast cancer screening is controversial. A 2013 Cochrane review stated that it is unclear if mammographic screening does more good or harm. A 2009 review for the US Preventive Services Task Force found evidence of

benefit in those 40 to 70 years of age, and the organization recommends screening every two years in women 50 to 74 years old. The medications tamoxifen or raloxifene may be used in an effort to prevent breast cancer in those who are at high risk of developing it. Surgical removal of both breasts is another useful preventative measure in some high risk women. In those who have been diagnosed with cancer, a number of treatments may be used, including surgery, radiation therapy, chemotherapy, and targeted therapy. Types of surgery vary from breast-conserving surgery to mastectomy. Breast reconstruction may take place at the time of surgery or at a later date. In those in whom the cancer has spread to other parts of the body, treatments are mostly aimed at improving quality of life and comfort. Outcomes for breast cancer vary depending on the cancer type, extent of disease, and person's age. Survival rates in the developed world are high, with between 80% and 90% of those in England and the United States alive for at least 5 years. In developing countries survival rates are poorer. Worldwide, breast cancer is the leading type of cancer in women, accounting for 25% of all cases. In 2012 it resulted in 1.68 million cases and 522,000 deaths. It is more common in developed countries and is more than 100 times more common in women than in men.

History of breast cancer : Breast cancer was the form of cancer most often described in ancient documents. Because autopsies were rare, cancers of the internal organs were essentially invisible to ancient medicine. Breast cancer, however, could be felt through the skin, and in its advanced state often developed into fungating lesions: the tumor would become necrotic (die from the inside, causing the tumor to appear to break up) and ulcerate through the skin, weeping fetid, dark fluid. The oldest description of cancer was discovered in Egypt and dates back to approximately 1600 BC. The Edwin Smith Papyrus describes 8 cases of tumors or ulcers of the breast that were treated by cauterization. The writing says about the disease, "There is no treatment." For centuries, physicians described similar cases in their practices, with the same conclusion. Ancient medicine, from the time of the Greeks through the 17th century, was based on humoralism, and thus believed that breast cancer was generally caused by imbalances in the fundamental fluids that controlled the body, especially an excess of black bile. Alternatively, patients often saw it as divine punishment. In the 18th century, a wide variety of medical explanations were proposed, including a lack of sexual activity, too much sexual activity, physical injuries to the breast, curdled breast milk, and various forms of lymphatic blockages, either internal or due to restrictive clothing. In the 19th century, the Scottish surgeon John Rodman said that fear of cancer caused cancer, and that this anxiety, learned by example from the mother, accounted for breast cancer's tendency to run in families. Although breast cancer was known in ancient times, it was uncommon until the 19th century, when improvements in sanitation and control of deadly infectious diseases resulted in dramatic increases in lifespan. Previously, most women had died too young to have developed breast cancer. Additionally, early and frequent childbearing and breastfeeding probably reduced the rate of breast cancer development in those women who did survive to middle age. Because ancient medicine believed that the cause was systemic, rather than local, and because surgery carried a high mortality rate, the preferred treatments tended to be

pharmacological rather than surgical. Herbal and mineral preparations, especially involving the poison arsenic, were relatively common. Mastectomy for breast cancer was performed at least as early as AD 548, when it was proposed by the court physician Aetios of Amida to Theodora. It was not until doctors achieved greater understanding of the circulatory system in the 17th century that they could link breast cancer's spread to the lymph nodes in the armpit. The French surgeon Jean Louis Petit (1674–1750) and later the Scottish surgeon Benjamin Bell (1749–1806) were the first to remove the lymph nodes, breast tissue, and underlying chest muscle. Their successful work was carried on by William Stewart Halsted who started performing radical mastectomies in 1882, helped greatly by advances in general surgical technology, such as aseptic technique and anesthesia. The Halsted radical mastectomy often involved removing both breasts, associated lymph nodes, and the underlying chest muscles. This often led to long-term pain and disability, but was seen as necessary in order to prevent the cancer from recurring. Before the advent of the Halsted radical mastectomy, 20-year survival rates were only 10%; Halsted's surgery raised that rate to 50%. Extending Halsted's work, Jerome Urban promoted superradical mastectomies, taking even more tissue, until 1963, when the ten-year survival rates proved equal to the less-damaging radical mastectomy. Radical mastectomies remained the standard of care in America until the 1970s, but in Europe, breast-sparing procedures, often followed radiation therapy, were generally adopted in the 1950s. One reason for this striking difference in approach may be the structure of the medical professions: European surgeons, descended from the barber surgeon, were held in less esteem than physicians; in America, the surgeon was the king of the medical profession. Additionally, there were far more European women surgeons: Less than one percent of American surgical oncologists were female, but some European breast cancer wards boasted a medical staff that was half female. American health insurance companies also paid surgeons more to perform radical mastectomies than they did to perform more intricate breast-sparing surgeries. Breast cancer staging systems were developed in the 1920s and 1930s. During the 1970s, a new understanding of metastasis led to perceiving cancer as a systemic illness as well as a localized one, and more sparing procedures were developed that proved equally effective. Modern chemotherapy developed after World War II. The French surgeon Bernard Peyrilhe (1737–1804) realized the first experimental transmission of cancer by injecting extracts of breast cancer into an animal. Prominent women who died of breast cancer include Anne of Austria, the mother of Louis XIV of France; Mary Washington, mother of George, and Rachel Carson, the environmentalist. The first case-controlled study on breast cancer epidemiology was done by Janet Lane-Claypon, who published a comparative study in 1926 of 500 breast cancer cases and 500 control patients of the same background and lifestyle for the British Ministry of Health. In the 1980s and 1990s, thousands of women who had successfully completed standard treatment then demanded and received high-dose bone marrow transplants, thinking this would lead to better long-term survival. However, it proved completely ineffective, and 15–20% of women died because of the brutal treatment. The 1995 reports from the Nurses' Health Study and the 2002 conclusions of the Women's Health

Initiative trial conclusively proved that hormone replacement therapy significantly increased the incidence of breast cancer.

Risk factors for breast cancer : A risk factor is anything that affects chance of getting a disease, such as cancer. Different cancers have different risk factors. For example, exposing skin to strong sunlight is a risk factor for skin cancer. Smoking is a risk factor for cancers of the lung, mouth, larynx (voice box), bladder, kidney, and several other organs. Having a risk factor, or even several, does not mean that you will get the disease. Most women who have one or more breast cancer risk factors never develop the disease, while many women with breast cancer have no apparent risk factors (other than being a woman and growing older). Even when a woman with risk factors develops breast cancer, it is hard to know just how much these factors might have contributed. Some risk factors, like a person's age or race, can't be changed. Others are linked to cancer-causing factors in the environment. Still others are related to personal behaviors, such as smoking, drinking, and diet. Some factors influence risk more than others, and risk for breast cancer can change over time, due to factors such as aging or lifestyle.

Gender : Simply being a woman is the main risk factor for developing breast cancer. Men can develop breast cancer, but this disease is about 100 times more common among women than men. This is probably because men have less of the female hormones estrogen and progesterone, which can promote breast cancer cell growth.

Aging : Over 80% of all female breast cancers occur among women aged 50+ years (after the menopause). About 1 out of 8 invasive breast cancers are found in women younger than 45, while about 2 of 3 invasive breast cancers are found in women age 55 or older.

Genetic risk factors : About 5% to 10% of breast cancer cases are thought to be hereditary, meaning that they result directly from gene defects (called mutations) inherited from a parent. If two close family members develop the disease, it does not necessarily mean they shared the genes that make them more vulnerable, because breast cancer is a relatively common cancer. The majority of breast cancers are not hereditary. Women who carry the BRCA1 and BRCA2 genes have a considerably higher risk of developing breast and/or ovarian cancer. These genes can be inherited. TP53, another gene, is also linked to greater breast cancer risk. In normal cells, these genes help prevent cancer by making proteins that keep the cells from growing abnormally. If inherited a mutated copy of either gene from a parent have a high risk of developing breast cancer during lifetime. Although in some families with BRCA1 mutations the lifetime risk of breast cancer is as high as 80%, on average this risk seems to be in the range of 55 to 65%. For BRCA2 mutations the risk is lower, around 45%. Breast cancers linked to these mutations occur more often in younger women and more often affect both breasts than cancers not linked to these mutations. Women with these inherited mutations also have an increased risk for developing other cancers, particularly ovarian cancer. In the United States BRCA mutations are more common in Jewish people of Ashkenazi (Eastern Europe) origin than in other racial and ethnic groups, but they can occur in anyone.

Changes in other genes : Other gene mutations can also lead to inherited breast cancers. These gene mutations are much rarer and often do not increase the risk of breast cancer as much as the BRCA genes. They are not frequent causes of inherited breast cancer.

Ataxia telangiectasia mutated (ATM) : The Ataxia telangiectasia mutated (ATM) gene normally helps repair damaged DNA. Inheriting abnormal copies of this gene causes the disease ataxia-telangiectasia. Inheriting mutated copy of this gene has been linked to a high rate of breast cancer in some families.

TP53 : The TP53 gene gives instructions for making a protein called p53 that helps stop the growth of abnormal cells. Inherited mutations of this gene cause Li-Fraumeni syndrome. People with this syndrome have an increased risk of developing breast cancer, as well as several other cancers such as leukemia, brain tumors, and sarcomas (cancer of bones or connective tissue). This is a rare cause of breast cancer.

CHEK2 : The Li-Fraumeni syndrome can also be caused by inherited mutations in the CHEK2 gene. Even when it does not cause this syndrome, it can increase breast cancer risk about twofold when it is mutated.

PTEN : The PTEN gene normally helps regulate cell growth. Inherited mutations in this gene can cause Cowden syndrome, a rare disorder in which people are at increased risk for both benign and malignant breast tumors, as well as growths in the digestive tract, thyroid, uterus, and ovaries. Defects in this gene can also cause a different syndrome called Bannayan-Riley-Ruvalcaba syndrome that is not thought to be linked to breast cancer risk. Recently, the syndromes caused by PTEN have been combined into one called PTEN Tumor Hamartoma Syndrome.

CDH1 : Inherited mutations in this gene cause hereditary diffuse gastric cancer, a syndrome in which people develop a rare type of stomach cancer at an early age. Women with mutations in this gene also have an increased risk of invasive lobular breast cancer.

STK11 : Defects in this gene can lead to Peutz-Jeghers syndrome. People with this disorder develop pigmented spots on their lips and in their mouths, polyps in the urinary and gastrointestinal tracts, and have an increased risk of many types of cancer, including breast cancer.

PALB2 : The PALB2 gene makes a protein that interacts with the protein made by the BRCA2 gene. Defects (mutations) in this gene can lead to an increased risk of breast cancer. It isn't yet clear if PALB2 gene mutations also increase the risk for ovarian cancer and male breast cancer.

Family history of breast cancer : Breast cancer risk is higher among women whose close blood relatives have this disease. Having one first-degree relative (mother, sister, or daughter) with breast cancer approximately doubles a woman's risk. Having 2 first-degree relatives increases her risk about 3-fold. The exact risk is not known, but women with a family history of breast cancer in a father or brother also have an increased risk of breast

cancer. Altogether, less than 15% of women with breast cancer have a family member with this disease. This means that most (over 85%) women who get breast cancer do not have a family history of this disease.

Personal history of breast cancer : A woman with cancer in one breast has a 3- to 4-fold increased risk of developing a new cancer in the other breast or in another part of the same breast. This is different from a recurrence (return) of the first cancer.

Race and ethnicity : Overall, white women are slightly more likely to develop breast cancer than are African-American women, but African-American women are more likely to die of this cancer. However, in women under 45 years of age, breast cancer is more common in African- American women. Asian, Hispanic, and Native-American women have a lower risk of developing and dying from breast cancer.

Dense breast tissue : Breasts are made up of fatty tissue, fibrous tissue, and glandular tissue. Someone is said to have dense breast tissue (as seen on a mammogram) when they have more glandular and fibrous tissue and less fatty tissue. Women with dense breasts on mammogram have a risk of breast cancer that is 1.2 to 2 times that of women with average breast density. Unfortunately, dense breast tissue can also make mammograms less accurate. A number of factors can affect breast density, such as age, menopausal status, certain medications (including menopausal hormone therapy), pregnancy, and genetics.

Estrogen exposure : Women who started having period are earlier or entered menopause later than usual has a higher risk of developing breast cancer. This is because their bodies have been exposed to estrogen for longer. Estrogen exposure begins when periods start, and drops dramatically during the menopause.

Obesity : Post-menopausal obese and overweight women may have a higher risk of developing breast cancer. Experts say that there are higher levels of estrogen in obese menopausal women, which may be the cause of the higher risk.

Height : Taller-than-average women have a slightly greater likelihood of developing breast cancer than shorter-than-average women. Experts are not sure why.

Certain benign breast conditions : Women diagnosed with certain benign breast conditions might have an increased risk of breast cancer. Some of these conditions are more closely linked to breast cancer risk than others. Doctors often divide benign breast conditions into 3 general groups, depending on how they affect this risk.

Non-proliferative lesions : These conditions are not associated with overgrowth of breast tissue. They do not seem to affect breast cancer risk, or if they do, it is to a very small extent. They include: Fibrosis and/or simple cysts (this used to be called fibrocystic disease or changes), Mild hyperplasia, Adenosis (non-sclerosing), Ductal ectasia, Phyllodes tumor (benign), A single papilloma, Fat necrosis, Periductal fibrosis, Squamous and apocrine metaplasia, Epithelial-related calcifications, Other benign tumors (lipoma, hamartoma, hemangioma, neurofibroma, adenomyoepithelioma). Mastitis (infection of the breast) is not a

lesion, but is a condition that can occur that does not increase the risk of breast cancer.

Proliferative lesions without atypia : These conditions show excessive growth of cells in the ducts or lobules of the breast tissue. They seem to raise a woman's risk of breast cancer slightly ($1\frac{1}{2}$ to 2 times normal). They include: Usual ductal hyperplasia (without atypia), Fibroadenoma, Sclerosing adenosis, Several papillomas (called papillomatosis), Radial scar.

Proliferative lesions with atypia : In these conditions, there is an overgrowth of cells in the ducts or lobules of the breast tissue, with some of the cells no longer appearing normal. They have a stronger effect on breast cancer risk, raising it $3\frac{1}{2}$ to 5 times higher than normal. These types of lesions include: Atypical ductal hyperplasia (ADH) and atypical lobular hyperplasia (ALH).

Women with a family history of breast cancer and either hyperplasia or atypical hyperplasia have an even higher risk of developing a breast cancer.

Lobular carcinoma in situ : In lobular carcinoma in situ (LCIS) cells that look like cancer cells are growing in the lobules of the milk-producing glands of the breast, but they do not grow through the wall of the lobules. LCIS (also called lobular neoplasia) is sometimes grouped with ductal carcinoma in situ (DCIS) as a non-invasive breast cancer, but it differs from DCIS in that it doesn't seem to become an invasive cancer if it isn't treated.

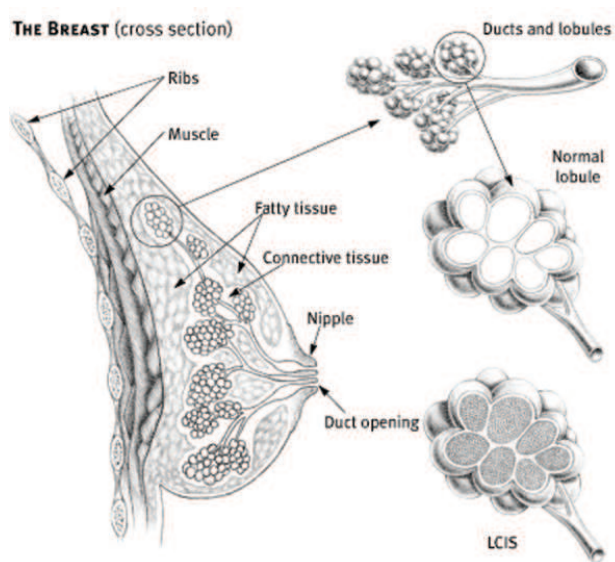


Fig: Lobular carcinoma in situ

Menstrual periods : Women who have had more menstrual cycles because they started menstruating early (before age 12) and/or went through menopause later (after age 55) have a slightly higher risk of breast cancer. The increase in risk may be due to a longer lifetime exposure to the hormones estrogen and progesterone.

Previous chest radiation : Women who, as children or young adults, had radiation

therapy to the chest area as treatment for another cancer (such as lymphoma) have a significantly increased risk for breast cancer. This varies with the patient's age when they had radiation. If chemotherapy was also given, it may have stopped ovarian hormone production for some time, lowering the risk. The risk of developing breast cancer from chest radiation is highest if the radiation was given during adolescence, when the breasts were still developing. Radiation treatment after age 40 does not seem to increase breast cancer risk.

Diethylstilbestrol exposure : From the 1940s through the 1960s some pregnant women were given the drug diethylstilbestrol (DES) because it was thought to lower their chances of miscarriage (losing the baby). These women have a slightly increased risk of developing breast cancer. Women whose mothers took DES during pregnancy may also have a slightly higher risk of breast cancer.

Lifestyle-related factors and breast cancer risk

Having children : Women who have had no children or who had their first child after the age of 30 years have a slightly higher breast cancer risk overall. Having many pregnancies and becoming pregnant at a young age reduce breast cancer risk overall. Still, the effect of pregnancy is different for different types of breast cancer. For a certain type of breast cancer known as triple-negative, pregnancy seems to increase risk.

Birth control:

Oral contraceptives : Studies have found that women using oral contraceptives (birth control pills) have a slightly greater risk of breast cancer than women who have never used them. This risk seems to go back to normal over time once the pills are stopped. Women who stopped using oral contraceptives more than 10 years ago do not appear to have any increased breast cancer risk. When thinking about using oral contraceptives, women should discuss their other risk factors for breast cancer with their health care team. Depot-medroxyprogesterone acetate (DMPA; Depo-Provera®) is an injectable form of progesterone that is given once every 3 months as birth control. A few studies have looked at the effect of DMPA on breast cancer risk. Women currently using DMPA seem to have an increase in risk, but the risk doesn't seem to be increased if this drug was used more than 5 years ago.

Hormone therapy after menopause : Hormone therapy with estrogen (often combined with progesterone) has been used for many years to help relieve symptoms of menopause and to help prevent osteoporosis (thinning of the bones). Earlier studies suggested it might have other health benefits as well, but these benefits have not been found in more recent, better designed studies. This treatment goes by many names, such as post-menopausal hormone therapy (PHT), hormone replacement therapy (HRT), and menopausal hormone therapy (MHT). There are 2 main types of hormone therapy. For women who still have a uterus (womb), doctors generally prescribe both estrogen and progesterone (known as combined hormone therapy or HT). Progesterone is needed because estrogen alone can increase the risk of cancer of the uterus. For women who no longer have a uterus (those who've had a

hysterectomy), estrogen alone can be prescribed. This is commonly known as estrogen replacement therapy (ERT) or just estrogen therapy (ET). Using combined hormone therapy after menopause increases the risk of getting breast cancer. It may also increase the chances of dying from breast cancer. This increase in risk can be seen with as little as 2 years of use. Combined HT also increases the likelihood that the cancer may be found at a more advanced stage. The increased risk from combined hormone therapy appears to apply only to current and recent users. A woman's breast cancer risk seems to return to that of the general population within 5 years of stopping combined treatment. The word bioidentical is sometimes used to describe versions of estrogen and progesterone with the same chemical structure as those found naturally in people. The use of these hormones has been marketed as a safe way to treat the symptoms of menopause. It is important to realize that although there are few studies comparing "bioidentical" or "natural" hormones to synthetic versions of hormones, there is no evidence that they are safer or more effective. The use of these bioidentical hormones should be assumed to have the same health risks as any other type of hormone therapy. The use of estrogen alone after menopause does not appear to increase the risk of developing breast cancer. In fact, some research has suggested that women who have previously had their uterus removed and who take estrogen actually have a lower risk of breast cancer. Women taking estrogen seem to have more problems with strokes and other blood clots, though. Also, when used long term (for more than 10 years), ET has been found to increase the risk of ovarian cancer in some studies. At this time there appear to be few strong reasons to use post-menopausal hormone therapy (either combined HT or ET), other than possibly for the short-term relief of menopausal symptoms. Along with the increased risk of breast cancer, combined HT also appears to increase the risk of heart disease, blood clots, and strokes. It does lower the risk of colorectal cancer and osteoporosis, but this must be weighed against possible harm, especially since there are other effective ways to prevent and treat osteoporosis. Although ET does not seem to increase breast cancer risk, it does increase the risk of blood clots and stroke. The decision to use hormone therapy after menopause should be made by a woman and her doctor after weighing the possible risks and benefits, based on the severity of her menopausal symptoms and the woman's other risk factors for heart disease, breast cancer, and osteoporosis. If a woman and her doctor decide to try hormones for symptoms of menopause, it is usually best to use it at the lowest dose needed to control symptoms and for as short a time as possible.

Breastfeeding : Some studies suggest that breastfeeding may slightly lower breast cancer risk, especially if it is continued for 1½ to 2 years. But this has been a difficult area to study, especially in countries such as the United States, where breastfeeding for this long is uncommon. One explanation for this possible effect may be that breastfeeding reduces a woman's total number of lifetime menstrual cycles (similar to starting menstrual periods at a later age or going through early menopause).

Drinking alcohol : The use of alcohol is clearly linked to an increased risk of developing

breast cancer. The risk increases with the amount of alcohol consumed. Compared with non-drinkers, women who consume 1 alcoholic drink a day have a very small increase in risk. Those who have 2 to 5 drinks daily have about 1½ times the risk of women who don't drink alcohol. Excessive alcohol consumption is also known to increase the risk of developing several other types of cancer.

Being overweight or obese : Being overweight or obese after menopause increases breast cancer risk. Before menopause your ovaries produce most of your estrogen, and fat tissue produces a small amount of estrogen. After menopause (when the ovaries stop making estrogen), most of a woman's estrogen comes from fat tissue. Having more fat tissue after menopause can increase your chance of getting breast cancer by raising estrogen levels. Also, women who are overweight tend to have higher blood insulin levels. Higher insulin levels have also been linked to some cancers, including breast cancer. But the connection between weight and breast cancer risk is complex. For example, the risk appears to be increased for women who gained weight as an adult but may not be increased among those who have been overweight since childhood. Also, excess fat in the waist area may affect risk more than the same amount of fat in the hips and thighs. Researchers believe that fat cells in various parts of the body have subtle differences that may explain this.

Physical activity : Evidence is growing that physical activity in the form of exercise reduces breast cancer risk. The main question is how much exercise is needed. In one study from the Women's Health Initiative, as little as 1.25 to 2.5 hours per week of brisk walking reduced a woman's risk by 18%. Walking 10 hours a week reduced the risk a little more.

Unclear factors:

Diet and vitamin intake : Many studies have looked for a link between what women eat and breast cancer risk, but so far the results have been conflicting. Some studies have indicated that diet may play a role, while others found no evidence that diet influences breast cancer risk. For example, a recent study found a higher risk of breast cancer in women who ate more red meat. Studies have also looked at vitamin levels, again with inconsistent results. Some studies actually found an increased risk of breast cancer in women with higher levels of certain nutrients. So far, no study has shown that taking vitamins reduces breast cancer risk. This is not to say that there is no point in eating a healthy diet. A diet low in fat, low in red meat and processed meat, and high in fruits and vegetables might have other health benefits. Most studies have found that breast cancer is less common in countries where the typical diet is low in total fat, low in polyunsaturated fat, and low in saturated fat. But many studies of women in the United States have not linked breast cancer risk to dietary fat intake. Researchers are still not sure how to explain this apparent disagreement. It may be at least partly due to the effect of diet on body weight (see below). Also, studies comparing diet and breast cancer risk in different countries are complicated by other differences (like activity level, intake of other nutrients, and genetic factors) that might also affect breast cancer risk. More research is needed to understand the effect of the types of fat eaten on breast cancer risk.

But it is clear that calories do count, and fat is a major source of calories. High-fat diets can lead to being overweight or obese, which is a breast cancer risk factor. A diet high in fat has also been shown to influence the risk of developing several other types of cancer, and intake of certain types of fat is clearly related to heart disease risk.

Chemicals in the environment : A great deal of research has been reported and more is being done to understand possible environmental influences on breast cancer risk. Compounds in the environment that have estrogen-like properties are of special interest. For example, substances found in some plastics, certain cosmetics and personal care products, pesticides (such as DDE), and PCBs (polychlorinated biphenyls) seem to have such properties. These could in theory affect breast cancer risk. This issue understandably invokes a great deal of public concern, but at this time research does not show a clear link between breast cancer risk and exposure to these substances. Unfortunately, studying such effects in humans is difficult. More research is needed to better define the possible health effects of these and similar substances.

Tobacco smoke : For a long time, studies found no link between cigarette smoking and breast cancer. In recent years though, more studies have found that long-term heavy smoking is linked to a higher risk of breast cancer. Some studies have found that the risk is highest in certain groups, such as women who started smoking when they were young. In 2009, the International Agency for Research on Cancer concluded that there is limited evidence that tobacco smoking causes breast cancer. An active focus of research is whether secondhand smoke increases the risk of breast cancer. Both mainstream and secondhand smoke contain chemicals that, in high concentrations, cause breast cancer in rodents. Chemicals in tobacco smoke reach breast tissue and are found in breast milk. The evidence on secondhand smoke and breast cancer risk in human studies is controversial, at least in part because the link between smoking and breast cancer hasn't been clear. One possible explanation for this is that tobacco smoke may have different effects on breast cancer risk in smokers and in those who are just exposed to smoke. A report from the California Environmental Protection Agency in 2005 concluded that the evidence about secondhand smoke and breast cancer is "consistent with a causal association" in younger, mainly premenopausal women. The 2006 US Surgeon General's report, *The Health Consequences of Involuntary Exposure to Tobacco Smoke*, concluded that there is "suggestive but not sufficient" evidence of a link at this point. In any case, this possible link to breast cancer is yet another reason to avoid secondhand smoke.

Night work : Several studies have suggested that women who work at night—for example, nurses on a night shift—may have an increased risk of developing breast cancer. This is a fairly recent finding, and more studies are looking at this issue. Some researchers think the effect may be due to changes in levels of melatonin, a hormone whose production is affected by the body's exposure to light, but other hormones are also being studied.

Controversial or disproven factors

Antiperspirants: Internet and e-mail rumors have suggested that chemicals in underarm antiperspirants are absorbed through the skin, interfere with lymph circulation, cause toxins to build up in the breast, and eventually lead to breast cancer. Based on the available evidence (including what we know about how the body works), there is little if any reason to believe that antiperspirants increase the risk of breast cancer. For more information about this, see our document *Antiperspirants and Breast Cancer Risk*.

Bras : Internet and e-mail rumors and at least one book have suggested that bras cause breast cancer by obstructing lymph flow. There is no good scientific or clinical basis for this claim, and a recent study of more than 1,500 women found no association of bra use with breast cancer risk.

Induced abortion : Several studies have provided very strong data that neither induced abortions nor spontaneous abortions (miscarriages) have an overall effect on the risk of breast cancer.

Breast implants : Several studies have found that breast implants do not increase the risk of breast cancer, although silicone breast implants can cause scar tissue to form in the breast. Implants make it harder to see breast tissue on standard mammograms, but additional x-ray pictures called implant displacement views can be used to examine the breast tissue more completely. Breast implants may be linked to a rare type of lymphoma called anaplastic large cell lymphoma. This lymphoma has rarely been found in the breast tissue around the implants. So far, though, there are too few cases to know if the risk of this lymphoma is really higher in women that have implants.

Signs and symptoms of breast cancer:

A symptom is only felt by the patient, and is described to the doctor or nurse, such as a headache or pain. A sign is something the patient and others can detect, for example, a rash or swelling. The first symptoms of breast cancer are usually an area of thickened tissue in the woman's breast, or a lump. The majority of lumps are not cancerous; however, women should get them checked by a health care professional. According to the National Health Service, UK, women who detect any of the following signs or symptoms should tell their doctor: Some of the possible early signs of breast cancer are

- A lump in a breast
- A pain in the armpits or breast that does not seem to be related to the woman's menstrual period
- Pitting or redness of the skin of the breast; like the skin of an orange
- A rash around (or on) one of the nipples
- A swelling (lump) in one of the armpits
- An area of thickened tissue in a breast

- One of the nipples has a discharge; sometimes it may contain blood
- The nipple changes in appearance; it may become sunken or inverted
- The size or the shape of the breast changes
- The nipple-skin or breast-skin may have started to peel, scale or flake.

ILLUSTRATION OF SYMPTOMS OF BREAST CANCER

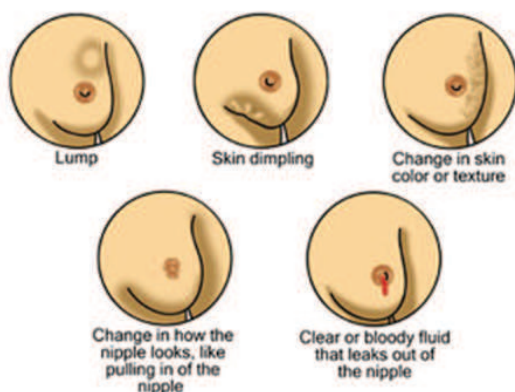


Fig: Some of the possible early signs of breast cancer

The following stages are used for breast cancer: The breast cancer stage is based on the results of testing that is done on the tumor and lymph nodes removed during surgery and other tests.

Stage 0 (carcinoma in situ): There are 3 types of breast carcinoma in situ:

- Ductal carcinoma in situ (DCIS) is a noninvasive condition in which abnormal cells are found in the lining of a breast duct. The abnormal cells have not spread outside the duct to other tissues in the breast. In some cases, DCIS may become invasive cancer and spread to other tissues. At this time, there is no way to know which lesions could become invasive.
- Lobular carcinoma in situ (LCIS) is a condition in which abnormal cells are found in the lobules of the breast. This condition seldom becomes invasive cancer. However, having LCIS in one breast increases the risk of developing breast cancer in either breast.
- Paget disease of the nipple is a condition in which abnormal cells are found in the nipple only.

Stage I : In stage I, cancer has formed. Stage I is divided into stages IA and IB.

- In stage IA, the tumor is 2 centimeters or smaller. Cancer has not spread outside the breast.

- In stage IB, small clusters of breast cancer cells (larger than 0.2 millimeter but not larger than 2 millimeters) are found in the lymph nodes and either:
 - ✓ no tumor is found in the breast; or
 - ✓ the tumor is 2 centimeters or smaller.

Stage II : Stage II is divided into stages IIA and IIB. In stage IIA:

- No tumor is found in the breast or the tumor is 2 centimeters or smaller. Cancer (larger than 2 millimeters) is found in 1 to 3 axillary lymph nodes or in the lymph nodes near the breastbone (found during a sentinel lymph node biopsy); or
- The tumor is larger than 2 centimeters but not larger than 5 centimeters. Cancer has not spread to the lymph nodes.

In stage IIB, the tumor is :

- Larger than 2 centimeters but not larger than 5 centimeters. Small clusters of breast cancer cells (larger than 0.2 millimeter but not larger than 2 millimeters) are found in the lymph nodes; or
- Larger than 2 centimeters but not larger than 5 centimeters. Cancer has spread to 1 to 3 axillary lymph nodes or to the lymph nodes near the breastbone (found during a sentinel lymph node biopsy); or
- Larger than 5 centimeters. Cancer has not spread to the lymph nodes.

In stage IIIA:

- No tumor is found in the breast or the tumor may be any size. Cancer is found in 4 to 9 axillary lymph nodes or in the lymph nodes near the breastbone (found during imaging tests or a physical exam); or
- The tumor is larger than 5 centimeters. Small clusters of breast cancer cells (larger than 0.2 millimeter but not larger than 2 millimeters) are found in the lymph nodes; or
- The tumor is larger than 5 centimeters. Cancer has spread to 1 to 3 axillary lymph nodes or to the lymph nodes near the breastbone (found during a sentinel lymph node biopsy).

In stage IIIB, the tumor may be any size and cancer has spread to the chest wall and/or to the skin of the breast and caused swelling or an ulcer. Also, cancer may have spread to:

- Up to 9 axillary lymph nodes; or
- The lymph nodes near the breastbone.

Cancer that has spread to the skin of the breast may also be inflammatory breast cancer.

In stage IIIC, no tumor is found in the breast or the tumor may be any size. Cancer may have spread to the skin of the breast and caused swelling or an ulcer and/or has spread to the chest wall. Also, cancer has spread to:

- 10 or more axillary lymph nodes; or

- Lymph nodes above or below the collarbone; or
- Axillary lymph nodes and lymph nodes near the breastbone.

Cancer that has spread to the skin of the breast may also be inflammatory breast cancer. For treatment, stage IIIC breast cancer is divided into operable and inoperable stage IIIC.

In stage IV, cancer has spread to other organs of the body, most often the bones, lungs, liver, or brain.

Diagnosing breast cancer : Women are usually diagnosed with breast cancer after a routine breast cancer screening, or after detecting certain signs and symptoms and seeing their doctor about them. If a woman detects any of the breast cancer signs and symptoms described above, she should speak to her doctor immediately. The doctor, often a primary care physician (general practitioner, GP) initially, will carry out a physical exam, and then refer the patient to a specialist if he/she thinks further assessment is needed. Most types of breast cancer are easy to diagnose by microscopic analysis of a sample—or biopsy—of the affected area of the breast. There are, however, rarer types of breast cancer that require specialized lab exams.

The two most commonly used screening methods, physical examination of the breasts by a healthcare provider and mammography, can offer an approximate likelihood that a lump is cancer, and may also detect some other lesions, such as a simple cyst. When these examinations are inconclusive, a healthcare provider can remove a sample of the fluid in the lump for microscopic analysis (a procedure known as fine needle aspiration, or fine needle aspiration and cytology—FNAC) to help establish the diagnosis. The needle aspiration may be performed in a healthcare provider's office or clinic using local anaesthetic if required. A finding of clear fluid makes the lump highly unlikely to be cancerous, but bloody fluid may be sent off for inspection under a microscope for cancerous cells. Together, physical examination of the breasts, mammography, and FNAC can be used to diagnose breast cancer with a good degree of accuracy. Other options for biopsy include a core biopsy or vacuum-assisted breast biopsy, which are procedures in which a section of the breast lump is removed; or an excisional biopsy, in which the entire lump is removed. Very often the results of physical examination by a healthcare provider, mammography, and additional tests that may be performed in special circumstances (such as imaging by ultrasound or MRI) are sufficient to warrant excisional biopsy as the definitive diagnostic and primary treatment method. Below are examples of diagnostic tests and procedures for breast cancer:

Breast exam - the physician will check both the patient's breasts, looking out for lumps and other possible abnormalities, such as inverted nipples, nipple discharge, or change in breast shape. The patient will be asked to sit/stand with her arms in different positions, such as above her head and by her sides.

X-ray (mammogram) - commonly used for breast cancer screening. If anything unusual is found, the doctor may order a diagnostic mammogram. Breast cancer screening has become a controversial subject over the last few years. Experts, professional bodies, and

patient groups cannot currently agree on when mammography screening should start and how often it should occur. Some say routine screening should start when the woman is 40 years old, others insist on 50 as the best age, and a few believe that only high-risk groups should have routine screening. In July, 2012, The American Medical Association said that women should be eligible for screening mammography from the age of 40, and it should be covered by insurance. In a Special Report in *The Lancet* (October 30th, 2012 issue), a panel of experts explained that breast cancer screening does reduce the risk of death from the disease. However, they added that it also creates more cases of false-positive results, where women end up having unnecessary biopsies and harmless tumors are surgically removed. Another study carried out by scientists at The Dartmouth Institute for Healthy Policy & Clinical Practice in Lebanon, N.H., and reported in the *New England Journal of Medicine* (November 2012 issue), found that mammograms do not reduce breast cancer death rates. A team from the University of Copenhagen reported that women who have false-positive mammogram outcomes may suffer long-lasting stress and anxiety, in some cases this can last up to three years. They published their findings in *Annals of Family Medicine* (March 2013 issue). Researchers from the Barbara Ann Karmanos Cancer Institute in Detroit, Michigan, found that breast cancer mortality was higher among older women whose time-lapses between their last mammogram and their breast cancer diagnosis were longer. They presented their findings at the American Association for Cancer Research (AACR) Annual Meeting 2013. Team leader, Michael S. Simon, M.D., M.P.H., said “We found that for women age 75 and older, a longer time interval between the last mammogram and the date of breast cancer diagnosis was associated with a greater chance for dying from breast cancer.”

2D combined with 3D mammograms - 3D mammograms, when used in collaboration with regular 2D mammograms were found to reduce the incidence of false positives, researchers from the University of Sydney’s School of Public Health, Australia, reported in *The Lancet Oncology*. The researchers screened 7,292 adult females, average age 58 years. Their initial screening was done using 2D mammograms, and then they underwent a combination of 2D and 3D mammograms. Professor Nehmat Houssami and team found 59 cancers in 57 patients. 66% of the cancers were detected in both 2D and combined 2D/3D screenings. However, 33% of them were only detected using the 2D plus 3D combination. The team also found that 2D plus 3D combination screenings were linked to a much lower number of false positives. When using just 2D screenings there were 141 false positives, compared to 73 using the 2D plus 3D combination. Prof. Houssami said “Although controversial, mammography screening is the only population-level early detection strategy that has been shown to reduce breast cancer mortality in randomized trials.

Ultrasound exam : A procedure in which high-energy sound waves (ultrasound) are bounced off internal tissues or organs and make echoes. The echoes form a picture of body tissues called a sonogram.

MRI (magnetic resonance imaging) : A procedure that uses a magnet, radio waves,

and a computer to make a series of detailed pictures of areas inside the body. This procedure is also called nuclear magnetic resonance imaging (NMRI).

Blood chemistry studies : A procedure in which a blood sample is checked to measure the amounts of certain substances released into the blood by organs and tissues in the body. An unusual (higher or lower than normal) amount of a substance can be a sign of disease in the organ or tissue that makes it.

Biopsy: The removal of cells or tissues so they can be viewed under a microscope by a pathologist to check for signs of cancer. If a lump in the breast is found, the doctor may need to remove a small piece of the lump. Four types of biopsies are as follows:

- **Excisional biopsy:** The removal of an entire lump of tissue.
- **Incisional biopsy:** The removal of part of a lump or a sample of tissue.
- **Core biopsy:** The removal of tissue using a wide needle.
- **Fine-needle aspiration (FNA) biopsy:** The removal of tissue or fluid, using a thin needle.

Prevention : Women may reduce their risk of breast cancer by maintaining a healthy weight, drinking less alcohol, being physically active and breastfeeding their children. These modifications might prevent 38% of breast cancers in the US, 42% in the UK, 28% in Brazil and 20% in China. The benefits with moderate exercise such as brisk walking are seen at all age groups including postmenopausal women. Marine omega-3 polyunsaturated fatty acids appear to reduce the risk. Removal of both breasts before any cancer has been diagnosed or any suspicious lump or other lesion has appeared (a procedure known as prophylactic bilateral mastectomy) may be considered in people with BRCA1 and BRCA2 mutations, which are associated with a substantially heightened risk for an eventual diagnosis of breast cancer. Evidence is not strong enough to support this procedure in anyone but those at the highest risk. BRCA testing is recommended in those with a high family risk after genetic counseling. It is not recommended routinely. This is because there are many different forms of changes in BRCA genes, ranging from harmless polymorphisms to obviously dangerous frameshift mutations. The effect of most of identifiable changes in the genes is uncertain. Testing in an average-risk person is particularly likely to return one of these indeterminate, useless results. It is unclear if removing the second breast in those who have breast cancer in one is beneficial. The selective estrogen receptor modulators (such as tamoxifen) reduce the risk of breast cancer but increase the risk of thromboembolism and endometrial cancer. There is no overall change in the risk of death. They are thus not recommended for the prevention of breast cancer in women at average risk but may be offered for those at high risk. The benefit of breast cancer reduction continues for at least five years after stopping a course of treatment with these medications.

Treatment of breast cancer : The management of breast cancer depends on various factors, including the stage of the cancer and the age of the patient.. Increasingly aggressive treatments are employed in accordance with the poorer the patient's prognosis and the

higher the risk of recurrence of the cancer following treatment. Breast cancer is usually treated with surgery, which may be followed by chemotherapy or radiation therapy, or both. A multidisciplinary approach is preferable. Hormone receptor-positive cancers are often treated with hormone-blocking therapy over courses of several years. Monoclonal antibodies, or other immune-modulating treatments, may be administered in certain cases of metastatic and other advanced stages of breast cancer. The main types of treatment for breast cancer are:

- Surgery
- Radiation therapy
- Chemotherapy
- Hormone therapy
- Targeted therapy
- Bone-directed therapy
- Nutrition and physical activity
- Follow-up care

Treatments can be classified into broad groups, based on how they work and when they are used. Local therapy is intended to treat a tumor at the site without affecting the rest of the body. Surgery and radiation therapy are examples of local therapies. Systemic therapy refers to drugs which can be given by mouth or directly into the bloodstream to reach cancer cells anywhere in the body. Chemotherapy, hormone therapy, and targeted therapy are systemic therapies.

Adjuvant and neoadjuvant therapy : Patients who have no detectable cancer after surgery are often given additional treatment to help keep the cancer from coming back. This is known as adjuvant therapy. Doctors believe that even in the early stages of breast cancer, cancer cells may break away from the primary breast tumor and begin to spread. These cells can't be felt on a physical exam or seen on x-rays or other imaging tests, and they cause no symptoms. But they can go on to become new tumors in nearby tissues, other organs, and bones. The goal of adjuvant therapy is to kill these hidden cells. Both systemic therapy (like chemotherapy, hormone therapy, and targeted therapy) and radiation can be used as adjuvant therapy. Most, but not all, patients benefit from adjuvant therapy. How much you might benefit depends on the stage and characteristics of the cancer and what type of surgery you had. Generally speaking, if the tumor is larger or the cancer has spread to lymph nodes, it is more likely to have spread through the bloodstream, and more likely to see a benefit. Some patients are given treatment, such as chemotherapy or hormone therapy, before surgery. The goal of this treatment is to shrink the tumor in the hope it will allow a less extensive operation to be done. This is called neoadjuvant therapy. Neoadjuvant therapy also lowers the chance of the cancer coming back later. Many patients who get neoadjuvant therapy will not need adjuvant therapy, or will not need as much.

Surgery : Surgery involves the physical removal of the tumor, typically along with some

of the surrounding tissue. One or more lymph nodes may be biopsied during the surgery; increasingly the lymph node sampling is performed by a sentinel lymph node biopsy. Standard surgeries include:

- **Mastectomy:** Removal of the whole breast.
- **Quadrantectomy:** Removal of one quarter of the breast.
- **Lumpectomy:** Removal of a small part of the breast.

Once the tumor has been removed, if the patient desires, breast reconstruction surgery, a type of plastic surgery, may then be performed to improve the aesthetic appearance of the treated site. Alternatively, women use breast prostheses to simulate a breast under clothing, or choose a flat chest. Nipple/areola prostheses can be used at any time following the mastectomy. Breast-conserving surgery (lumpectomy or partial mastectomy) can often be used for early-stage breast cancers. But in some women, it can result in breasts of different sizes and/or shapes. For larger tumors, it might not even be possible, and a mastectomy might be needed instead. Some doctors address this problem by combining cancer surgery and plastic surgery techniques, known as oncoplastic surgery. This typically involves reshaping the breast at the time of the initial surgery, and may mean operating on the other breast as well to make them more symmetrical. This approach is still fairly new, and not all doctors are comfortable with it.

Hormone blocking therapy : Some breast cancers require estrogen to continue growing. They can be identified by the presence of estrogen receptors (ER+) and progesterone receptors (PR+) on their surface (sometimes referred to together as hormone receptors). These ER+ cancers can be treated with drugs that either block the receptors, e.g. tamoxifen, or alternatively block the production of estrogen with an aromatase inhibitor, e.g. anastrozole or letrozole. The use of tamoxifen is recommended for 10 years. Aromatase inhibitors, however, are only suitable for post-menopausal patients. This is because the active aromatase in postmenopausal women is different from the prevalent form in premenopausal women, and therefore these agents are ineffective in inhibiting the predominant aromatase of premenopausal women.

New chemotherapy drugs : Advanced breast cancers are often hard to treat, so researchers are always looking for newer drugs. A drug class has been developed that targets cancers caused by BRCA mutations. This class of drugs is called PARP inhibitors and they have shown promise in clinical trials treating breast, ovarian, and prostate cancers that had spread and were resistant to other treatments.

Targeted therapies : Targeted therapies are a group of newer drugs that specifically take advantage of gene changes in cells that cause cancer.

Drugs that target HER2 : A number of drugs that target HER2 are currently in use, including trastuzumab (Herceptin), pertuzumab (Perjeta), ado-trastuzumab emtansine (Kadcyla), and lapatinib (Tykerb). Other drugs are being developed and tested.

Anti-angiogenesis drugs : For cancers to grow, blood vessels must develop to nourish

the cancer cells. This process is called angiogenesis. Looking at angiogenesis in breast cancer specimens can help predict prognosis. Some studies have found that breast cancers surrounded by many new, small blood vessels are likely to be more aggressive. More research is needed to confirm this. Bevacizumab (Avastin) is an example of anti-angiogenesis drug. Although bevacizumab turned out to not be very helpful in the treatment of advanced breast cancer, this approach still may prove useful in breast cancer treatment. Several other anti-angiogenesis drugs are being tested in clinical trials.

Other targeted drugs : Everolimus (Afinitor) is a targeted therapy drug that seems to help hormone therapy drugs work better. It is approved to be given with exemestane (Aromasin) to treat advanced hormone receptor-positive breast cancer in post-menopausal women. It has also been studied with other hormone therapy drugs and for treatment of earlier stage breast cancer. In one study, letrozole plus everolimus worked better than letrozole alone in shrinking breast tumors before surgery. It also seemed to help in treating advanced hormone receptor-positive breast cancer when added to tamoxifen. Everolimus is also being studied in combination with chemotherapy and the targeted drug trastuzumab. Other drugs like everolimus are also being studied. Other potential targets for new breast cancer drugs have been identified in recent years. Drugs based on these targets are now being studied, but most are still in the early stages of clinical trials.

Bisphosphonates : Bisphosphonates are drugs that are used to help strengthen and reduce the risk of fractures in bones that have been weakened by metastatic breast cancer. Examples include pamidronate (Aredia) and zoledronic acid (Zometa). Some studies have suggested that zoledronic acid may help other systemic therapies, like hormone treatment and chemo work better. In one study of women being treated with chemo before surgery, tumors in the women getting zoledronic acid with chemo shrank more than those in the women treated with chemo alone. Other studies have looked at the effect of giving zoledronic acid with other adjuvant treatments (like chemo or hormone therapy). So far, the results have been mixed. Some studies have shown that this approach helped lower the risk of the cancer coming back, but others did not. The results of one study linked the use of these drugs with adjuvant chemo with an increased risk of breast cancer recurrence in younger women. Overall, the data does not support making bisphosphonates part of standard therapy for early-stage breast cancer.

Denosumab : Denosumab (Xgeva, Prolia) can also be used to help strengthen and reduce the risk of fractures in bones that have been weakened by metastatic breast cancer. It is being studied to see if it can help adjuvant treatments work better.

Vitamin D : A recent study found that women with early-stage breast cancer who were vitamin D deficient were more likely to have their cancer recur in a distant part of the body and had a poorer outlook. More research is needed to confirm this finding. It is not yet clear if taking vitamin D supplements would be helpful. Still, you might want to talk to your doctor about testing your vitamin D level to see if it is in the healthy range.

Monoclonal antibodies : Trastuzumab, a monoclonal antibody to HER2 (a cell receptor that is especially active in some breast cancer cells), has improved the 5-year disease free survival of stage 1–3 HER2-positive breast cancers to about 87% (overall survival 95%). When stimulated by certain growth factors, HER2 causes cellular growth and division; in the absence of stimulation by the growth factor, the cell will normally stop growing. Between 25% and 30% of breast cancers over express the HER2 gene or its protein product, and overexpression of HER2 in breast cancer is associated with increased disease recurrence and worse prognosis. When trastuzumab binds to the HER2 in breast cancer cells that over express the receptor, trastuzumab prevents growth factors from being able to bind to and stimulate the receptors, effectively blocking the growth of the cancer cells. Trastuzumab, however, is very expensive, and its use may cause serious side effects (approximately 2% of patients who receive it suffer significant heart damage). Further, trastuzumab is only effective in patients with HER2 amplification/over expression.

Radiation : Radiotherapy is given after surgery to the region of the tumor bed and regional lymph nodes, to destroy microscopic tumor cells that may have escaped surgery. It may also have a beneficial effect on tumor microenvironment. Radiation therapy can be delivered as external beam radiotherapy or as brachytherapy (internal radiotherapy). Conventionally radiotherapy is given after the operation for breast cancer. Radiation can also be given at the time of operation on the breast cancer- intraoperatively. The largest randomised trial to test this approach was the TAR-GIT-A Trial which found that targeted intraoperative radiotherapy was equally effective at 4-years as the usual several weeks’ of whole breast external beam radiotherapy. Radiation can reduce the risk of recurrence by 50–66% (1/2 – 2/3 reduction of risk) when delivered in the correct dose and is considered essential when breast cancer is treated by removing only the lump (Lumpectomy or Wide local excision).

Breast cancer survival rates by stage:

Stage	5-year relative survival rate
0	100%
I	98 - 100%
II	93%
III	72%
IV	10 - 22%

The stage of the breast cancer is the most important component of traditional classification methods of breast cancer, because it has a greater effect on the prognosis than the other considerations. Staging takes into consideration size, local involvement, lymph node status and whether metastatic disease is present. The higher the stage at diagnosis, the poorer the prognosis. The stage is raised by the invasiveness of disease to lymph nodes, chest wall, skin or beyond, and the aggressiveness of the cancer cells. The stage is lowered by the presence of cancer-free zones and close-to-normal cell behaviour (grading). Size is not a

factor in staging unless the cancer is invasive. For example, Ductal Carcinoma In Situ (DCIS) involving the entire breast will still be stage zero and consequently an excellent prognosis with a 10-year disease free survival of about 98% - 100%.

- Stage 1 cancer (DCIS, LCIS) have an excellent prognosis and are generally treated with lumpectomy and sometimes radiation. HER2+ cancers should be treated with the trastuzumab (Herceptin) regime. Chemotherapy is uncommon for other types of stage 1 cancers.
- Stage 2 and 3 cancers with a progressively poorer prognosis and greater risk of recurrence are generally treated with surgery (lumpectomy or mastectomy with or without lymph node removal), chemotherapy (plus trastuzumab for HER2+ cancers) and sometimes radiation (particularly following large cancers, multiple positive nodes or lumpectomy).
- Stage 4, metastatic cancer, (i.e. spread to distant sites) has poor prognosis and is managed by various combination of all treatments from surgery, radiation, chemotherapy and targeted therapies. 10-year survival rate is 5% without treatment and 10-22% with optimal treatment.

The breast cancer grade is assessed by comparison of the breast cancer cells to normal breast cells. The closer to normal the cancer cells are, the slower their growth and the better the prognosis. If cells are not well differentiated, they will appear immature, will divide more rapidly, and will tend to spread. Well differentiated is given a grade of 1, moderate is grade 2, while poor or undifferentiated is given a higher grade of 3 or 4 (depending upon the scale used).

The presence of estrogen and progesterone receptors in the cancer cell is important in guiding treatment. Those who do not test positive for these specific receptors will not be able to respond to hormone therapy, and this can affect their chance of survival depending upon what treatment options remain, the exact type of the cancer, and how advanced the disease is. In addition to hormone receptors, there are other cell surface proteins that may affect prognosis and treatment. HER2 status directs the course of treatment. Patients whose cancer cells are positive for HER2 have more aggressive disease and may be treated with the 'targeted therapy', trastuzumab (Herceptin), a monoclonal antibody that targets this protein and improves the prognosis significantly. Younger women tend to have a poorer prognosis than post-menopausal women due to several factors. Their breasts may change with their menstrual cycles, they may be nursing infants, and they may be unaware of changes in their breasts. Therefore, younger women are usually at a more advanced stage when diagnosed. There may also be biologic factors contributing to a higher risk of disease recurrence for younger women with breast cancer. High mammographic breast density, which is a marker of increased risk of developing breast cancer, may not mean an increased risk of death among breast cancer patients, according to a 2012 report of a study involving 9232 women by the National Cancer Institute (NCI). Since breast cancer in males is usually detected at later stages, outcomes are typically worse.

Survival rates are often used by doctors as a standard way of discussing a person's prognosis (outlook). Some patients with breast cancer may want to know the survival statistics for people in similar situations, while others may not find the numbers helpful, or may even not want to know them. The 5-year observed survival rate refers to the percentage of patients who live at least 5 years after being diagnosed with cancer. Many of these patients live much longer than 5 years after diagnosis. A relative survival rate (like the numbers below) compares the observed survival with what would be expected for people without the cancer. This helps to correct for the deaths caused by something besides cancer and is a more accurate way to describe the effect of cancer on survival. In order to get 5-year survival rates, doctors have to look at people who were treated at least 5 years ago. Improvements in treatment since then may result in a more favorable outlook for people now being diagnosed with breast cancer. Survival rates are often based on previous outcomes of large numbers of people who had the disease, but they cannot predict what will happen in any particular person's case. Many other factors may affect a person's outlook, such as age and health, the presence of hormone receptors on the cancer cells, the treatment received, and how well the cancer responds to treatment. The available statistics do not divide survival rates by all of the substages, such as IA and IB. The rates for these substages are likely to be close to the rate for the overall stage. For example, the survival rate for stage IA is likely to be slightly higher than that listed for stage I, while the survival rate for stage IB would be expected to be slightly lower. It is also important to realize that these statistics are based on the stage of the cancer when it was first diagnosed. These do not apply to cancers that later come back or spread, for example. The rates below come from the National Cancer Institute's SEER database. They are based on the previous version of AJCC staging. In that version stage II also included patients that would now be considered stage IB.

Certain factors affect prognosis (chance of recovery) and treatment options: The prognosis and treatment options depend on the following:

- The stage of the cancer (the size of the tumor and whether it is in the breast only or has spread to lymph nodes or other places in the body).
- The type of breast cancer.
- Estrogen receptor and progesterone receptor levels in the tumor tissue.
- Human epidermal growth factor type 2 receptor (HER2/neu) levels in the tumor tissue.
- Whether the tumor tissue is triple-negative (cells that do not have estrogen receptors, progesterone receptors, or high levels of HER2/neu).
- How fast the tumor is growing.
- How likely the tumor is to recur (come back).
- A woman's age, general health, and menopausal status (whether a woman is still having menstrual periods).
- Whether the cancer has just been diagnosed or has recurred (come back).

What happens after treatment for breast cancer? Completing treatment can be both stressful and exciting. When cancer comes back after treatment, it is called recurrence. This is a very common concern in people who have had cancer. It may help to know that many cancer survivors have learned to live with this uncertainty and are leading full lives. For other people, the cancer may never go away completely. These people may get regular treatments with chemotherapy, radiation therapy, or other treatments to try to help keep the cancer in check. Learning to live with cancer that does not go away can be difficult and very stressful. It has its own type of uncertainty.

Follow-up care : When treatment ends, doctors will still want to watch closely. It is very important to go to all of follow-up appointments. During these visits, doctors will ask questions about any problems you may have and may do exams and lab tests or x-rays and scans to look for signs of cancer or treatment side effects. Almost any cancer treatment can have side effects. Some may last for a few weeks to months, but others can last the rest of life. This is the time for talk to cancer care team about any changes or problems you notice and any questions or concerns you have. At first, follow-up appointments will probably be scheduled for every 3 to 6 months. The longer you have been free of cancer, the less often the appointments are needed. After 5 years, they are typically done about once a year. If you had breast-conserving surgery, you will get a mammogram about 6 months after surgery and radiation are completed, and then at least every year. Women who had a mastectomy should continue to have yearly mammograms on the remaining breast. If you are taking tamoxifen or toremifene, you should have pelvic exams every year because these drugs can increase risk of uterine cancer. This risk is highest in women who have gone through menopause. Be sure to tell doctor right away about any abnormal vaginal bleeding, such as vaginal bleeding or spotting after menopause, bleeding or spotting between periods, or a change in periods. Although this is usually caused by a non-cancerous condition, it can also be the first sign of uterine cancer. If you are taking an aromatase inhibitor for early stage breast cancer, doctor will want to monitor bone health and may consider testing bone density. Other tests such as blood tumor marker studies, blood tests of liver function, CTs, bone scans, and chest x-rays are not a standard part of follow-up because they don't help a woman treated with breast cancer live longer. But they will be done (as indicated) if you have symptoms or physical exam findings that suggest that the cancer has recurred. These and other tests may also be done as part of evaluating new treatments by clinical trials.

If symptoms, exams, or tests suggest a recurrence, imaging tests such as an x-ray, CT scan, PET scan, MRI scan, bone scan, and/or a biopsy may be done. Doctor may also look for circulating tumor cells in the blood or measure levels of blood tumor markers such as CA-15-3, CA 27-29, or CEA. The blood levels of tumor markers go up in some women if their cancer has spread to bones or other organs such as the liver. They are not elevated in all women with recurrence, so they aren't always helpful. If they are elevated, doctor might use them to monitor the results of therapy. If cancer does recur, treatment will depend on the location of the cancer and what treatments you had before. It may mean surgery, radiation therapy, hormone

therapy, chemotherapy, targeted therapy, or some combination of these. It is also important to keep health insurance. Tests and doctor visits cost a lot, and even though no one wants to think of their cancer coming back, this could happen.

Recent research on breast cancer : Treatments are constantly evaluated in randomized, controlled trials, to evaluate and compare individual drugs, combinations of drugs, and surgical and radiation techniques. Investigations include new types of targeted therapy as well as cancer vaccines. The latest research is reported annually at scientific meetings such as that of the American Society of Clinical Oncology, San Antonio Breast Cancer Symposium, and the St. Gallen Oncology Conference in St. Gallen, Switzerland. These studies are reviewed by professional societies and other organizations, and formulated into guidelines for specific treatment groups and risk category.

Breast cancer cell lines : A considerable part of the current knowledge on breast carcinomas is based on in vivo and in vitro studies performed with cell lines derived from breast cancers. These provide an unlimited source of homogenous self-replicating material, free of contaminating stromal cells, and often easily cultured in simple standard media. The first breast cancer cell line described, BT-20, was established in 1958. Since then, and despite sustained work in this area, the number of permanent lines obtained has been strikingly low (about 100). Indeed, attempts to culture breast cancer cell lines from primary tumors have been largely unsuccessful. This poor efficiency was often due to technical difficulties associated with the extraction of viable tumor cells from their surrounding stroma. Most of the available breast cancer cell lines issued from metastatic tumors, mainly from pleural effusions. Effusions provided generally large numbers of dissociated, viable tumor cells with little or no contamination by fibroblasts and other tumor stroma cells. Many of the currently used BCC lines were established in the late 1970s. A very few of them, namely MCF-7, T-47D, and MDA-MB-231, account for more than two-thirds of all abstracts reporting studies on mentioned breast cancer cell lines, as concluded from a Medline-based survey.

Molecular markers

Transcription factors : NFAT transcription factors are implicated in breast cancer, more specifically in the process of cell motility at the basis of metastasis formation. Indeed NFAT1 (NFATC2) and NFAT5 are pro-invasive and pro-migratory in breast carcinoma and NFAT3 (NFATc4) is an inhibitor of cell motility. NFAT1 regulates the expression of the TWEAKR and its ligand TWEAK with the Lipocalin 2 to increase breast cancer cell invasion and NFAT3 inhibits Lipocalin 2 expression to blunt the cell invasion.

Metabolic markers : Clinically, the most useful metabolic markers in breast cancer are the estrogen and progesterone receptors that are used to predict response to hormone therapy. New or potentially new markers for breast cancer include BRCA1 and BRCA2 to identify patients at high risk of developing breast cancer, HER-2 and SCD1 for predicting response to therapeutic regimens, and urokinase plasminogen activator, PA1-1 and SCD1 for assessing prognosis.

LEUKEMIA

The word Leukemia comes from the Greek 'leukos' which means "white" and 'aima' which means "blood" and refers to excess white blood cells in the body. Leukemia is a group of cancers that usually begins in the blood or bone marrow (which produces blood cells) results in high numbers of abnormal white blood cells. These white blood cells are not fully developed; these cells are commonly known as blast or leukemia cells. Person suffers with leukemia produced abnormal blood cells, generally leukocytes (white blood cells). Symptoms may include bleeding and bruising problems, feeling very tired, and an increased risk of infections. These symptoms occur due to a lack of normal blood cells. The DNA of immature blood cells, mainly white cells, becomes damaged in some way. This abnormality causes the blood cells to grow and divide uncontrollably. Normal blood cells die after a while and are replaced by new cells which are produced in the bone marrow. The abnormal blood cells do not die so easily, and accumulate, occupying more and more space. As more and more space is occupied by these faulty blood cells there is less and less space for the normal cells - and the sufferer becomes ill. Quite simply, the bad cells crowd out the good cells in the blood.

Today, the number of children seen to be affected by this disease is very high. The reason can be attributed to the lifestyle changes that have come along. Another probable reason could be that the percentage of mothers who breastfeed their children has come down drastically. During breastfeeding, the child's immune system comes across antibodies from the mother's body and evolves to respond to infections after birth. However, children who haven't been breastfed are more prone to develop leukemia, as they do not confront microbes in their early years.

Industrialized nations are more susceptible to leukemia because people residing in such nations are constantly coming in contact with chemicals, such as high levels of benzene and formaldehyde (at workplaces). Exposure to radiation via atomic bomb explosion or medical treatments such as chemotherapy and high amount of pesticides is also risk factors that can lead to leukemia. People suffering from Down syndrome are also prone to this blood disease. The oldest and primary treatment for leukemia was arsenic. In the 18th century, Thomas Fowler created a solution comprising arsenic trioxide and potassium bicarbonate, and called it Fowler's solution. This solution became a standard remedy to treat Hodgkin's disease, anemia, and leukemia. However, in the early 20th century, arsenic was replaced by radiation therapy. Radiation therapy was found to be very beneficial in curing leukemia. The American Cancer Society says that the early radiologists used the skin of their own hands to test the strength of radiation from the radiotherapy machines in search of the appropriate doses (to get pink skin after radiation), which was to be the right amount for the treatment. Sadly, most of them came down with leukemia. It was only after World War II that chemotherapy came in as a treatment for leukemia. Further, in the 1940s more and more new treatments such as aminopterin and 6-mercaptopurine came. The discovery of DNA has helped understand the

detailed mechanisms of cancer and the reason why it occurs. Bone marrow transplants are known to be the best cure for leukemia today. Genetic analysis is expected to open new doors towards the treatment and cure of leukemia in the future. Leukemia at a glance is a cancer of the blood cells and although the cause of this disease is not known, the risk factors that lead to it have been identified. People need to be careful about the amount of radiation and chemicals they are being exposed to.

Discovery of Leukemia

The credit for its discovery goes to the ancient Greeks, who recognized this blood disease way back in the 4th or 5th century BC. However, it was first officially diagnosed by Rudolf Virchow and John Hughes Bennett in Edinburgh in 1845. Further, in the 19th century, several European physicians noticed that quite a few of their patients suffered from abnormally high levels of white blood cells. They called this condition 'weisses blut', which meant white blood. In the year of 1900, leukemia was viewed as a family of diseases as opposed to a single disease. By 1947 Boston pathologist Sidney Farber believed from past experiments that aminopterin, a folic acid mimic, could potentially cure leukemia in children. The majority of the children with all who were tested showed signs of improvement in their bone marrow, but none of them were actually cured. In 1962, Emil J. Freireich Jr. and Emil Frei III used combination chemotherapy to attempt to cure leukemia. The tests were successful with some patients surviving long after the tests. In 1970, it was confirmed that leukemia could be cured, and by the 1980s and 1990s the patients cured were around 70%.

Types of Leukemia

There are different types of cancers that affect patients around the world. One such cancer is leukemia. Clinically and pathologically, leukemia is subdivided into a variety of large groups.

The first division of leukemia is according to its acute and chronic form.

The first division of leukemia is according to its acute and chronic form.

Based on Proliferation

- **Acute Leukemia** - this type of leukemia is characterized by the rapid growth of immature blood cells. This crowding makes the bone marrow unable to produce healthy blood cells. Acute forms of leukemia can occur in children and young adults. Immediate treatment is required due to the rapid progression and accumulation of the malignant cells, which then spill over into the bloodstream and spread to other organs of the body. If left untreated, the patient will die within months or even weeks.
- **Chronic leukemia** - chronic leukemia is distinguished by the excessive build up of relatively mature, but still abnormal, blood cells. Typically taking months to years to progress, the cells are produced at a higher rate than normal cells, resulting in many abnormal white blood cells in the blood. Chronic leukemia mostly occurs in older people, but can theoretically occur in any age group. Chronic forms are sometimes

monitored for some time before treatment to make maximum effectiveness of therapy.

Based on kind of blood cell affected

The disease is classified according to the type of abnormal cells found in the blood

- Lymphocytic leukemia – commonly known as lymphoblastic leukemia, it occurs when the B cell lymphocytes turn become abnormal and lead to leukemia.
- Myelogenous Leukemia - Myeloid leukemia or myelogenous leukemia is a type of cancer that occurs in red blood cells and sometimes affects the white blood cells as well as platelets.

Based on the above two groups

The above two groups forms the bases of the four main kinds of leukemia's. These types of leukemia may further have more subcategories, but the main types are as follows:

- **Acute lymphocytic leukemia (ALL)** - also known as acute lymphoblastic leukemia or (ALL) is the most common type of leukemia in young children. This disease also affects adults, especially at age 65 and older. This type is a cancer of the white blood cells, characterized by the overproduction and continuous multiplication of malignant and immature white blood in the bone marrow.
- **Acute myelogenous leukemia (AML)** - Also known as acute myeloid leukemia (AML) occurs more commonly in adults than in children. This was previously called acute nonlymphocytic leukemia.
- **Chronic lymphocytic leukemia (CLL)** - CLL most often affects adults over the age of 55. It sometimes occurs in younger adults, children are never affected by CLL.
- **Chronic myelogenous leukemia (CML)** - CML occurs mainly in adults. A very small number of children also develop this disease. This type is characterized by increased and unregulated clonal production of predominantly myeloid cells in the bone marrow.
- **Hairy cell leukemia (HCL)** - This leukemia is generally a subset of chronic lymphocytic leukemia. It mostly affects adult men and is incurable. However, there are treatments that help control the proliferation of this cancer.
- **Large granular lymphocytic leukemia** - The T-cells or NK cells are involved and is a very rare type of leukemia. This rare cancer is not very aggressive; therefore treatment is successful in most cases.
- **T-cell prolymphocytic leukemia (T-PLL)** - This is a very rare form of leukemia that is really aggressive in nature. It affects adults and involves mature T cells. This is very difficult to treat and survival rate is poor.
- **Adult T-cell leukemia** - The human T-lymphotropic virus (HTLV) can affect the CD4+ T-cells. This causes them to replicate and proliferate abnormally leading to adult T-cell leukemia.

Causes of leukemia

The exact cause of leukemia is still unknown. Different types of leukemia's have different causes. Both inherited and environmental (non-inherited) factors are believed to be involved. These are

- Smoking
- Artificial ionizing radiation
- **Viruses** - HTLV-1 (human T-lymphotropic virus) and HIV (human immunodeficiency virus)
- Benzene and some petrochemicals
- Alkylating chemotherapy agents used in previous cancers
- Maternal fetal transmission (rare)
- Hair dyes
- **Genetic predisposition**-some studies indicated that some people have a higher risk of developing leukemia because of a single gene or multiple genes.
- **Down syndrome**-people with Down syndrome have a significantly higher risk of developing leukemia, compared to people who do not have Down syndrome. Some studies have shown that people with certain chromosomal abnormalities may have a higher risk.
- **Electromagnetic energy** - there is not enough evidence to show that ELF magnetic (not electric) fields that exist currently might cause leukemia. The IARC (International Agency for Research on Cancer) says that studies which indicate there is a risk tend to be biased and unreliable.

Symptoms

Leukemia usually starts in the bone marrow, where white blood cells are produced. In Leukemia, there is an overproduction of white blood cells that are abnormal or “stuck” in an early stage of the maturation process. These leukemia cells are non functional and are unable to do the job of healthy, mature blood cells. In addition, their presence in the bone marrow crowds and prevents the ability of other normal blood forming cells to do their jobs. This leads to the signs and symptoms of leukemia. Signs and symptoms of acute leukemia (fast growing) may be similar to the flu and come on suddenly within days or weeks. In the early stages of chronic leukemia (slow growing), many people have few or no symptoms. Signs and symptoms usually develop gradually and people will complain that they just do not feel well. The most common symptoms of leukemia are vague and non-specific. As a result, they are often explained away by the patient as “coming down with something” or getting “run down.” The most common symptoms of leukemia include:

- **Blood clotting is poor** - The abnormal production of leukemia cells prevents the bone marrow from making adequate numbers of healthy blood cells, such as platelets. Platelets

are fragments of cells that clump together and stop or slow bleeding when an injury occurs to a blood vessel. When there are insufficient platelets or “thrombocytopenia”, bleeding may occur in the form of nosebleeds, heavy menstrual bleeding, bleeding gums, and bruises or bleed easily and heal slowly. The patient may develop a small red to purple spot on the body, caused by minor hemorrhage (known as petechiae).

- **Feeling weak, tired or generally unwell** - In most cases, this is caused by a decreased number of red blood cells in the bloodstream, or anemia. This prevents adequate oxygen being transported to our tissues and muscles, leaving our body feeling fatigued and weak.
- **Affected immune system** - The patient’s white blood cells, which are crucial for fighting off infection, may be suppressed or not working properly. The patient may experience frequent infections, or his immune system may attack other good body cells. Some patients experience frequent infection, ranging from infected tonsils, sores in the mouth, or diarrhea to life-threatening pneumonia or opportunistic infections.
- **Anemia** - As the shortage of good red blood cells grows the patient may suffer from anemia - this may lead to difficult or labored respiration (dyspnea) and pallor (skin has a pale color caused by illness).
- **Bone and Joint Pain** - Bone and joint pain is most common in areas where there is a large amount of bone marrow, such as the pelvis (hips) or breastbone (sternum). This is caused by the crowding of the marrow with excessive numbers of abnormal white blood cells.
- **Enlarged Lymph Nodes** - Sometimes, leukemia cells can accumulate in the lymph nodes and cause them to become swollen and tender. Some patients experience nausea or a feeling of fullness due to an enlarged liver and spleen; this can result in unintentional weight loss. Blasts affected by the disease may come together and become swollen in the liver or in the lymph nodes causing pain and leading to nausea.
- **Unexplained Fevers** - In some cases, leukemia cells can cause body to release chemicals that stimulate our brain to raise our body temperature. Fevers can also be caused by an infection.
- **Abdominal Discomfort** - Abnormal white blood cells can also collect in the liver and spleen causing our abdomen to swell and become uncomfortable. This type of swelling can also decrease our appetite, or make you feel “full” early.
- **Headaches and Other Neurological Complaints** - If the leukemic cells invade the central nervous system, then neurological symptoms (Headaches) can occur and other neurologic symptoms such as seizures, dizziness, visual changes and nausea and vomiting may occur. This type of central nervous system (CNS) involvement is most common in acute lymphocytic leukemia (ALL).

Precaution - All these symptoms could be due to other illnesses. A diagnosis of leukemia

can only be confirmed after medical tests are carried out. Leukemia cannot be diagnosed based on the presence of signs and symptoms alone. There are a number of tests and procedures that must be completed to confirm a suspected case of leukemia. It is very important to note that these signs and symptoms can also be caused by many other, non-cancerous conditions. If you are worried about any symptoms you are experiencing, you should always seek assistance from a qualified healthcare provider.

Treatments of leukemia : Leukemia is not a single disease. Instead, the term leukemia refers to a number of related cancers that start in the blood-forming cells of the bone marrow. There are both acute and chronic forms of leukemia, each with many subtypes that vary in their response to treatment. In addition, children with leukemia have special needs that are best met by care in pediatric cancer centers. Such centers have trained medical professionals whose sole purpose is to address the unique concerns of children. Leukemia treatment plans often are personalized and geared toward each individual patient. In general, there are five major approaches to the treatment of leukemia:

- Chemotherapy to kill leukemia cells using strong anti-cancer drugs;
- Interferon therapy to slow the reproduction of leukemia cells and promote the immune systems.
- Radiation therapy to kill cancer cells by exposure to high-energy radiation;
- Stem cell transplantation (SCT) to enable treatment with high doses of chemotherapy and radiation therapy; and
- Surgery to remove an enlarged spleen or to install a venous access device (large plastic tube) to give medications and withdraw blood samples.

Chemotherapy : Chemotherapy is the use of anti-cancer drugs. The aim is to destroy all cancer cells while doing the least possible damage to normal cells. The drugs work by stopping cancer cells from growing and reproducing. Chemotherapy drugs are usually taken intravenously, that is, they are injected or infused into a vein. To avoid having repeated injections, a long-lasting catheter in a vein is usually used. The drugs can then be added to a fluid drip attached to the catheter. Some chemotherapy is in tablet form or given as injections under the skin. Chronic lymphocytic leukaemia is usually treated in this way. For some people, chemotherapy for acute leukaemia will mean spending several weeks in hospital. Others may be able to stay at home but will need regular hospital visits for check-ups and further treatments. Three different phases of chemotherapy are used to treat acute leukaemia – induction therapy, consolidation therapy and maintenance therapy.

Induction therapy : Treatment begins with induction therapy, which usually lasts four to six weeks. This is a phase of intense treatment aimed at destroying as many abnormal white blood cells as possible. The aim is to effect a remission. After remission, people with acute lymphocytic leukaemia have extra treatment. As microscopic collections of leukaemic cells may have spread to the spinal fluid, chemotherapy drugs are injected directly into the fluid

around our spine. In children, radiotherapy is often not used because it may affect growth and development.

Consolidation therapy : After remission, more chemotherapy can be used to try to stop the cancer coming back (called a relapse). The goal of this second phase of treatment is to destroy any cancer cells that may have survived the first treatment. A number of different chemotherapy drugs are usually used. This is in case the leukaemia cells are resistant to any one drug.

Maintenance therapy : Maintenance therapy is the final stage of treatment for acute lymphocytic leukaemia in children. It's given over a longer period of time (two to three years), but its aim is the same: to destroy remaining cancer cells. In most of the acute myeloid leukaemias, the role and duration of maintenance chemotherapy is still being studied. In many people it isn't currently used. In general, this phase of treatment isn't as intense as the first two phases. It may sometimes be replaced by stem cell (bone marrow) transplantation after high-dose chemotherapy.

The most important effect of chemotherapy is that it kills leukaemia cells. However, it may have side effects. Normal cells are better able to renew themselves after chemotherapy than leukaemia cells, but some normal cells which multiply rapidly (such as hair cells and normal blood cells) may be affected by chemotherapy. Reactions vary with different drugs, with different people and from one course of treatment to the next. Doses of chemotherapy which are moderate, such as those used for treatment of chronic leukaemia, usually cause few side effects. The most common side effects are nausea and vomiting, feeling off-colour and tired, hair loss, diarrhoea, constipation and a sore mouth. If normal blood cells are affected, we may also have problems with infection and bleeding. Imatinib (Glivec) is being increasingly used to treat chronic myeloid leukaemia. It's sometimes added to chemotherapy to treat a type of acute lymphocytic leukaemia. Its side effects include mild feelings of sickness, diarrhoea, leg aches and cramps, rashes, and swelling around the eyes. All-trans retinoic acid, used to treat a type of acute myeloid leukaemia, may cause headaches, bone pain and dry skin. Remember that side effects can be prevented or controlled.

Stem cell (or bone marrow) transplantation : This treatment allows higher doses of chemotherapy than usual. This may increase our chance of being cured. It can be exhausting and has significant risks. Newer forms of transplantation (called mini allografts or reduced-intensity allografts) use lower doses of chemotherapy. They attempt to use the immune system to fight the leukaemia. Some younger patients with acute leukaemia in remission this treatment greatly increases the chance of long-term remission and cure. Stem cell transplantation is rarely used as the first treatment for children with acute lymphocytic leukaemia because chemotherapy usually works very well. Stem cells grow in bone marrow (the soft tissue inside bones). They're immature cells, from which essential new cells for the body grow. High doses of chemotherapy can harm stem cells.

Interferon Therapy to Treat Leukemia : Interferons are a class of proteins that are

released by virus-infected cells. They help normal cells to make antiviral proteins. Interferons also help the body to reduce leukemia cell proliferation (growth and reproduction), while strengthening the body's immune response. Interferon-alpha (INF- α) is a type of interferon that frequently is used to treat leukemia. In addition, based on a patient's response to INF- α , a physician can better predict the anticipated length of survival. Interferon-alpha can be given by injection into a vein, a muscle, or under the skin—although subcutaneous (under the skin) injection is the customary route. INF- α usually is offered to all newly diagnosed patients who are not candidates for stem cell transplantation. Often IFN- α is started at a low dose (e.g., 3 MIU daily), with gradual increases over time. Unfortunately, though, this drug is not without side effects. Possible IFN-related complaints include fevers, chills, muscle aches, bone pain, headaches, concentration difficulties, fatigue, nausea, vomiting, and general flu-like symptoms when starting the drug. Such symptoms usually last for 1 to 2 weeks, but may be lessened by drugs such as acetaminophen. Side effects recur if the INF- α dosage is increased, but they are temporary and usually improve after INF- α therapy is completed.

Recovery and follow-up care : Doctors may want to examine every 3 months for the first year after treatment, every 6 months between the second and fifth years of treatment, and once a year after that. They'll examine and ask about any symptoms and will answer any questions. Doctor may order other tests or scans if they think they're needed.

Life after treatment : After treatment for leukaemia likely to face several changes in life. For some people these changes may be short term. Other changes may be permanent and difficult to cope with. Most people find they need information and support about how to best deal with their situation.

HODGKIN LYMPHOMA

Hodgkin's lymphoma formerly known as Hodgkin's lymphoma or Hodgkin's disease or Charlie Hodgkin's disease is a cancer of the lymphatic system. It was named after Dr. Thomas Hodgkin, who first described abnormalities in the lymph system in 1832. The lymphatic system forms part of the immune system. It contains specialized white blood cells called lymphocytes that help protect the body from infection and disease. In Hodgkin's lymphoma, cells in the lymphatic system grow abnormally and may spread beyond the lymphatic system. These abnormal lymphocytes, called lymphoma cells, form collections of cancer cells called tumors, in lymph nodes (sometimes known as 'glands') and other parts of the body. The first sign of Hodgkin disease is often an enlarged lymph node. The disease may be found because of a swollen lymph node in the neck, chest, or other areas. The disease can spread to nearby lymph nodes. Later it may spread to the lungs, liver, or bone marrow. When white blood cells collect around the abnormal cells, the lymph node that contains abnormal cells becomes swollen. Abnormal cells may spread through the lymph vessels or blood vessels to other parts of the body. Although normal cells die when they get old or damaged, abnormal cells don't die. Hodgkin lymphoma is distinguished from all other types of lymphoma because of the presence of a special kind of cancer cell called a Reed-Sternberg cell. Hodgkin lymphoma can occur at any age but it is most common in adolescents and young adults. Over a third of all cases diagnosed are between the ages of 15 and 30. Hodgkin lymphoma occurs more frequently in males than in females.

Cells in different parts of the body work in different ways, but they all repair and reproduce themselves in the same way. Normally, cells divide in an orderly and controlled way. But if for some reason the process gets out of control, the cells continue to divide. This may lead to too many immature white blood cells in the blood or bone marrow. A lump or tumor may then develop in one or more groups of lymph nodes. Lymphoma cells generally start to grow in lymph nodes. As there are lymph nodes and lymph vessels throughout the body, Hodgkin lymphoma can start in any part of the body. The most common place for it to start is in the lymph nodes in the neck. The next most common places are the lymph nodes such as under the arms (axilla), in the chest and in the groin. Hodgkin lymphoma may affect a group of lymph nodes in just one area of the body. However, it's common for lymphoma to be found in lymph nodes in more than one area of the body, as lymphoma cells can sometimes spread through the lymphatic system. Lymphoma cells can also go into the bloodstream, which may carry them to other organs. When the cells reach a new area, they may go on dividing and form a new tumor. Hodgkin lymphoma can also occur in body organs. In some people it can affect the spleen, liver, lungs or bone marrow. Hodgkin lymphoma is diagnosed by a full blood count (FBC) and a bone marrow biopsy/examination.

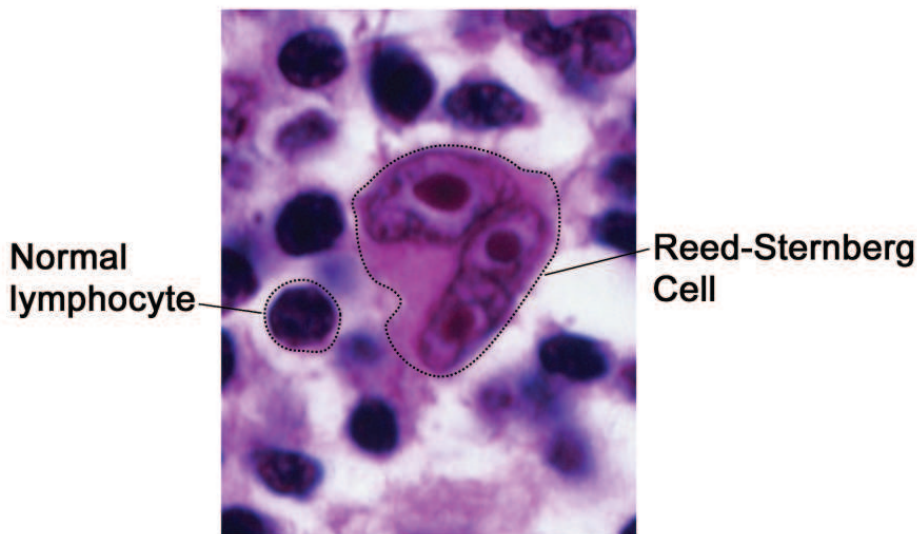


Fig: Normal lymphocyte and Reed-Sternberg lymphocyte

The lymphatic system : The lymph system (also known as the lymphatic system) is part of the immune system, which helps fight infections and some other diseases. It also helps fluids move in the body. The lymphatic system is made up of lymphatic organs such as bone marrow, the tonsils, the thymus, the spleen and lymph nodes (sometimes called lymph glands). The lymph nodes throughout the body are connected by a network of tiny lymphatic tubes (ducts). There are lymph nodes in many places in the body but they're mainly found in the neck, armpit and groin. Their number varies from one part of the body to another, and there are very few in some parts. However, under our arm there may be 20-50 nodes. Circulating the lymphatic vessels is milky-looking fluid called lymph, which contains lymphocytes. Lymphocytes are white blood cells. They are an essential part of the body's defense against infection and disease. As it circulates, lymph passes through the lymph nodes, which filter out the bacteria that cause infection. So if we have a sore throat, we may notice that the lymph nodes in our neck get larger. This is a sign that our body is fighting the infection. The lymph system is composed mainly of:

- **Lymph vessels :** Lymphatic vessels are small tubes, similar to blood vessels. The lymph system has a network of lymph vessels. Lymph vessels branch into all the tissues of the body through which lymph travels to different parts of the lymph system.
- **Lymph** a clear fluid that travels through the lymph system, carrying waste products and excess fluid from tissues, as well as lymphocytes and other immune system cells. Lymph contains white blood cells, especially lymphocytes such as B cells and T cells.
- **Lymph nodes :** Lymph vessels are connected to small, round organs called lymph nodes. Groups of lymph nodes are found in the neck, armpits, chest, abdomen, and groin. Lymph nodes store white blood cells. They trap and remove harmful substances

that may be in lymph. Other parts of the lymph system include the tonsils, thymus, and spleen. Lymph tissue is also found in other parts of the body including the stomach, skin, and small intestine.

Lymphoid tissue is made up mainly of cells called lymphocytes, a type of white blood cell. All lymphocytes develop in the bone marrow from immature cells called stem cells. The two major types of lymphocytes are B lymphocytes (B cells) and T lymphocytes (T cells).

- **B lymphocytes** : Lymphocytes that mature in the bone marrow or lymphatic organs are called B-cells. B cells help protect the body from germs (bacteria and viruses) by making proteins called antibodies. The antibodies attach to the germs, marking them for destruction by other parts of the immune system. Almost all cases of Hodgkin disease start in B lymphocytes.
- **T lymphocytes** : Lymphocytes that mature in the thymus gland (behind the breastbone) are called T-cells. There are several types of T cells, and each has a special job. Some T cells directly destroy certain kinds of bacteria or cells infected with viruses or fungi. Other types of T cells play a role in either boosting or slowing the activity of other immune system cells.

The major sites of lymphoid tissue are:

Lymph nodes : Lymph nodes are bean-sized collections of lymphocytes and other immune system cells throughout the body, including inside the chest, abdomen, and pelvis. They can sometimes be felt as small lumps under the skin in the neck, under the arms, and in the groin. Lymph nodes are connected to each other by a system of lymphatic vessels. Lymph nodes get bigger when they fight an infection. Lymph nodes that grow because of infection are called reactive or hyperplastic nodes. These often hurt when they are touched. People with sore throats or colds might have enlarged neck lymph nodes. An enlarged lymph node is not always a sign of a serious problem, but it can be a sign of Hodgkin disease.

Spleen : The spleen is an organ under the lower part of the rib cage on the left side of the body. The spleen makes lymphocytes and other immune system cells to help fight infection. It also stores healthy blood cells and filters out damaged blood cells, bacteria, and cell waste.

Bone marrow : The bone marrow is the spongy tissue inside certain bones, which is where new white blood cells (including some lymphocytes), red blood cells, and platelets are made.

Thymus : The thymus is a small organ behind the upper part of the breastbone and in front of the heart. It is important in the development of T lymphocytes.

Adenoids and tonsils : These are collections of lymphoid tissue in the back of the throat. They help make antibodies against germs that are breathed in or swallowed.

Digestive tract : The stomach, intestines, and many other organs also have lymphoid tissue. Lymphoid tissue is in many parts of the body, Hodgkin disease can start almost anywhere. Most often it starts in lymph nodes in the upper part of the body. The most

common sites are in the chest, in the neck, or under the arms. Hodgkin disease most often spreads through the lymph vessels in a stepwise fashion from lymph node to lymph node. Rarely, and late in the disease, it can invade the bloodstream and spread to other parts of the body, including the liver, lungs, and/or bone marrow.

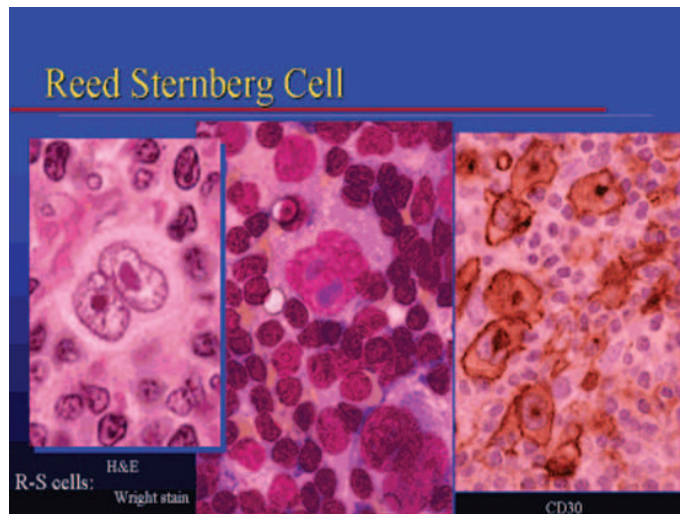


Fig: Reed-Sternberg cells

History of Hodgkin's Lymphoma :

Hodgkin's lymphoma is a cancer of the lymphatic system that affects the B-lymphocytes, causing them to accumulate in the lymph nodes. The cancer is named after Thomas Hodgkin who first described abnormalities in the lymphatic system in 1832. However, Hodgkin did note that the earliest description of the disease may have been given by Marcello Malpighi in 1666. While working at Guy's Hospital, Hodgkin studied seven patients who had enlarged but painless lymph nodes. One of these individuals was a patient of Robert Carswell, whose report of the patient included many illustrations that were used as an aid in early descriptions of the condition. Hodgkin wrote a report on the seven patients entitled "On some morbid appearances of the absorbent glands and spleen," which was presented to the Medical and Chirurgical Society in London in 1832 and then published in the society's journal, "Medical-Chirurgical Society Transactions."

In 1856, Samuel Wilks reported on a further set of patients with the same disease described by Hodgkin and in 1965 he published a paper entitled "Cases of Lardaceous Disease and Some Allied Affections with Remarks." In that paper, Wilks referred to the condition as "Hodgkin's disease," in honor of Hodgkin's previous contribution to the subject. In 1872, Langhans described the features of Hodgkin's lymphoma at the microscopic level and Carl Sternberg and Dorothy Reed first referred to the cytogenic characteristics of the cancerous cells in 1898 and 1902, respectively. Today these cells are called Reed-Sternberg cells. Tissue specimens from Hodgkin's seven patients remained at Guy's Hospital for a number of years.

Nearly 100 years after Hodgkin's initial publication, histopathologic reexamination confirmed Hodgkin's lymphoma in only three of seven of these patients. The remaining cases included non-Hodgkin lymphoma, tuberculosis, and syphilis. Hodgkin's lymphoma was one of the first cancers which could be treated using radiation therapy and, later, it was one of the first to be treated by combination chemotherapy. Treatments for Hodgkin's lymphoma were developed as early on as 1894, when Fowler's solution, which was a medicine containing arsenic was used. In 1932, Chevalier and Bernard described the use of radiation therapy to treat the condition, mainly for palliative purposes. Radiotherapy started to prove successful as a treatment in the mid-forties. In 1963, a combination of chemotherapy agents referred to as MOMP was developed, which consisted of cyclophosphamide, vincristine, methotrexate, and prednisone. Another drug regimen was introduced in 1987 called EBVP (epirubicin, bleomycin, vinblastine, prednisone). In 1992, The German Hodgkin's Study Group designed the BEACOPP regimen which involved the use of seven chemotherapy agents, namely bleomycin, etoposide, adriamycin, cyclophosphamide, oncovin, procarbazine and prednisone.

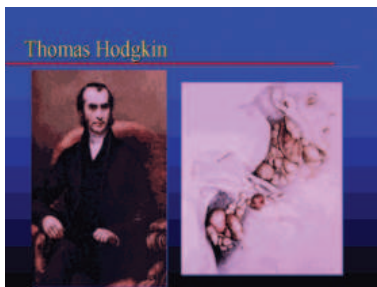


Fig: Thomas Hodgkin

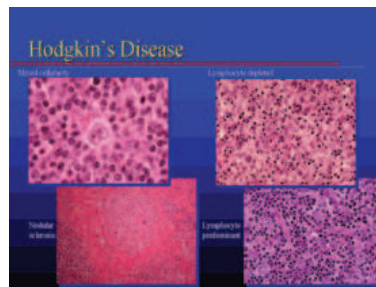


Fig: Hodgkin's Lymphoma

Causes of Hodgkin lymphoma:

The causes of Hodgkin lymphoma remain unknown but it is thought that damage of the genes that normally controls blood cell development. It is thought that alterations in the immune system may play a role in some cases. Infection with Epstein-Barr virus (EBV), that causes glandular fever, is believed to have a connection with the disease. There are a number of factors that can lead to an increased chance of developing lymphoma. These include a weakened immune system, (immunosuppressed) due to an inherited immune deficiency disease, HIV infection or drugs taken to prevent rejection of a transplanted organ. In the UK, Hodgkin lymphoma is most common in younger people aged 20-34 and older people aged 70-79, but it can occur at any age. Having a member of the family with Hodgkin lymphoma may slightly increase someone's risk of getting it. It's not yet known whether this is caused by an inherited faulty gene or because families may have similar lifestyle factors that affect their risk. The identical twin of someone with Hodgkin lymphoma has a slightly higher risk than other members of the family, but since Hodgkin lymphoma is not common, this risk is very small. Hodgkin lymphoma is not infectious and can't be passed on to other people. Fewer siblings, early birth order, single-family homes, and fewer playmates are associated with an increased risk of developing Hodgkin lymphoma possibly due to a lack of exposure

to bacterial and viral infections at an early age.

Risk factors for Hodgkin disease : A risk factor is anything that affects our chance of getting a disease such as cancer. Different cancers have different risk factors. Some cancer risk factors, like smoking, can be changed. Others, like a person's age or family history, can't be changed. Scientists have found a few risk factors that make a person more likely to develop Hodgkin disease (although it's not always clear why these factors increase risk). But having a risk factor, or even several, does not mean that definitely get the disease. Many people who get the disease may have few or no known risk factors. Even if a person with Hodgkin disease has one or more risk factors, it is often very hard to know how much these factors might have contributed to the lymphoma.

Epstein-Barr virus infection/mononucleosis : People who have had infectious mononucleosis (sometimes called mono for short), an infection caused by the Epstein-Barr virus (EBV), have an increased risk of Hodgkin disease. Although the risk is higher than for people who have not had mono, the overall risk is still very small. The exact role of EBV in the development of Hodgkin disease is not clear. Many people are infected with EBV, but very few develop Hodgkin disease. Parts of the virus are found in Reed-Sternberg cells in about 1 out of 3 patients with Hodgkin disease. But the other people with Hodgkin disease have no signs of EBV in their cancer cells.

Age : People of any age can be diagnosed with Hodgkin disease, but it is most common in early adulthood (ages 15 to 40, especially in a person's 20s) and in late adulthood (after age 55).

Gender : Hodgkin disease occurs slightly more often in males than in females.

Geography : Hodgkin disease is most common in the United States, Canada, and northern Europe, and is least common in Asian countries.

Family history : Brothers and sisters of young people with this disease have a higher risk for Hodgkin disease. The risk is very high for an identical twin of a person with Hodgkin disease. But a family link is still uncommon – most people with Hodgkin disease do not have a family history of it. It's not clear why family history might increase risk. It might be because family members have similar childhood exposures to certain infections (such as Epstein-Barr virus), inherited gene changes that make them more likely to get Hodgkin disease, or some combination of these factors.

Socioeconomic status : The risk of Hodgkin disease is greater in people with a higher socioeconomic background. The reason for this is not clear. One theory is that children from more affluent families might be exposed to some type of infection (such as Epstein-Barr virus) later in life than children from less affluent families, which might somehow increase their risk.

HIV infection : The risk of Hodgkin disease is increased in people infected with HIV, the virus that causes AIDS.

Types of Hodgkin disease : Different types of Hodgkin disease are classified by how they look under the microscope. This is important because types of Hodgkin disease may grow and spread differently and may be treated differently. The main two types are:

- Classic Hodgkin disease
- Nodular lymphocyte predominant Hodgkin disease

All types of Hodgkin disease are malignant (cancerous) because as they grow they can invade and destroy normal tissue and spread to other tissues.

Classic Hodgkin disease : Classical Hodgkin's lymphoma is the more common type of this disease. It can be broken down further into subtypes. People diagnosed with classical Hodgkin's lymphoma have large, abnormal cells called Reed-Sternberg cells in their lymph nodes. Classic Hodgkin disease (HD) accounts for about 95% of all cases of Hodgkin disease in developed countries. The cancer cells in classic HD are called Reed-Sternberg cells. These cells are usually an abnormal type of B lymphocyte. Reed-Sternberg cells are much larger than normal lymphocytes and also look different from the cells of non-Hodgkin lymphomas and other cancers. The enlarged lymph nodes in classic HD usually have a small number of Reed-Sternberg cells and a large number of surrounding normal immune cells. It is mainly these other immune cells that account for the enlarged lymph nodes.

Subtypes of classical Hodgkin's lymphoma include:

- Nodular sclerosis Hodgkin's lymphoma
- Mixed cellularity Hodgkin's lymphoma
- Lymphocyte-depleted Hodgkin's lymphoma
- Lymphocyte-rich classical Hodgkin's lymphoma

Nodular sclerosis Hodgkin disease : This is the most common type of Hodgkin disease in developed countries, accounting for about 60% to 80% of cases. It is most common in teens and young adults, but it can occur in people of any age. It tends to start in lymph nodes in the neck or chest.

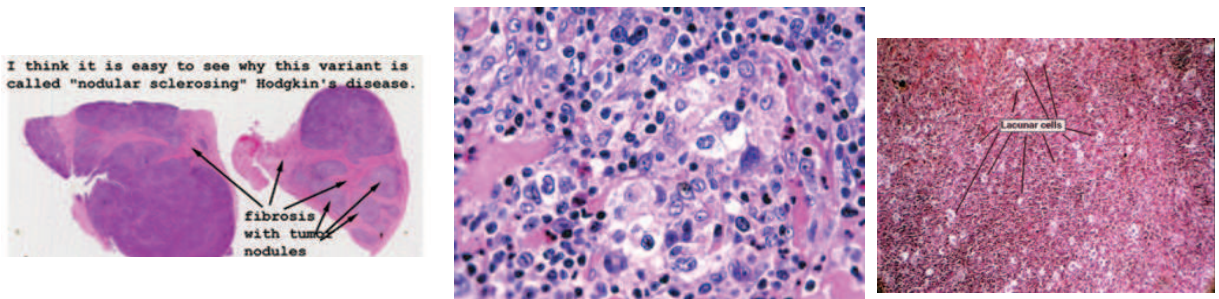


Fig: Nodular sclerosis Hodgkin disease

Mixed cellularity Hodgkin disease : This is the second most common type (15% to 30%) and is seen mostly in older adults (although it can occur at any age). It can start in any lymph node but most often occurs in the upper half of the body.

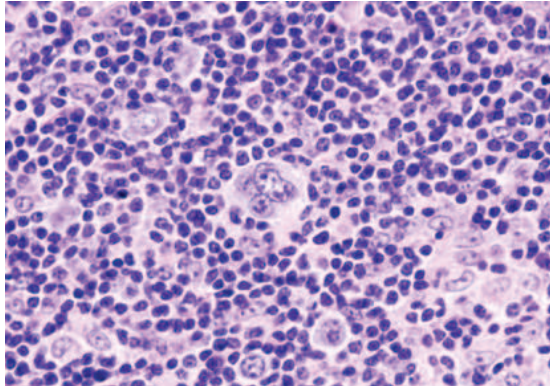


Fig: Mixed cellularity Hodgkin disease

Lymphocyte-rich Hodgkin disease : This subtype accounts for about 5% of Hodgkin disease cases. It usually occurs in the upper half of the body and is rarely found in more than a few lymph nodes.

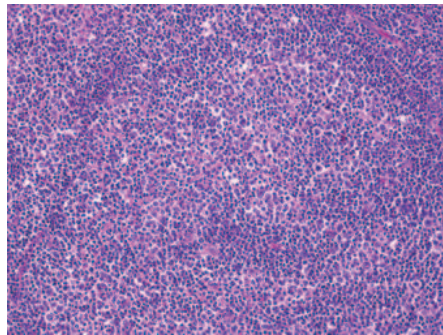


Fig: Lymphocyte-rich Hodgkin disease

Lymphocyte-depleted Hodgkin disease : This is the least common form of Hodgkin disease, making up less than 1% of cases. It is seen mainly in older people. The disease is more likely to be advanced when first found, in lymph nodes in the abdomen as well as in the spleen, liver, and bone marrow.

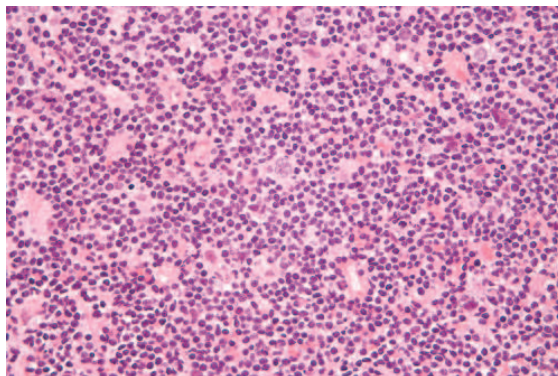


Fig: Lymphocyte-depleted Hodgkin disease

Nodular lymphocyte predominant Hodgkin disease : Nodular lymphocyte predominant Hodgkin disease (NLPHD) accounts for about 5% of Hodgkin disease. It is more common in older people but can occur in young people. The main difference between this type and classical Hodgkin lymphoma is that in the nodular lymphocyte predominant type there are very few Reed-Sternberg cells. But there are other abnormal cells that doctors call popcorn cells (because they look like popcorn). This type of Hodgkin lymphoma is often only in one group of lymph nodes when it is diagnosed (localised disease). It tends to be slower growing than classical Hodgkin lymphoma and the treatment is different. NLPHD usually starts in lymph nodes in the neck and under the arm. It can occur in people of any age, and is more common in men than in women.

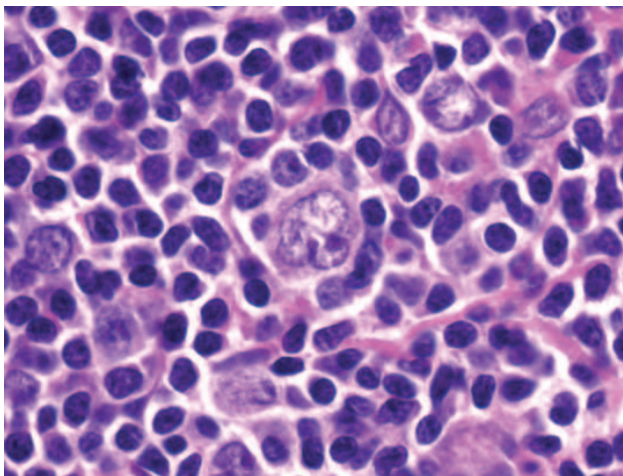


Fig: Nodular lymphocyte predominant Hodgkin disease

Staging determined for Hodgkin's lymphoma : Staging tests can show the stage (extent) of disease, such as whether lymphoma cells are found in more than one group of lymph nodes. Lymphoma cells usually spread from one group of lymph nodes to the next. For example, Hodgkin lymphoma that starts in lymph nodes in the neck may spread first to lymph nodes above the collarbones, and then to lymph nodes under the arms and within the chest. In time, lymphoma cells can invade blood vessels and spread to almost any other part of the body. For example, they can spread to the liver, lungs, bone, and bone marrow. Staging tests may include:

CT scan : Doctor may order a CT scan of our neck, chest, abdomen, and pelvis. An x-ray machine linked to a computer will take a series of detailed pictures of these areas. We'll receive contrast material by mouth and by injection into a blood vessel in our arm or hand. The contrast material makes swollen lymph nodes and other abnormal areas easier to see. The pictures can show whether Hodgkin lymphoma has spread.

PET scan : Doctor may use a PET scan to find Hodgkin lymphoma that has spread. We'll receive an injection of a small amount of radioactive sugar. A machine makes computerized

pictures of cells in our body that have taken up the radioactive sugar due to lymphoma cells take up sugar faster than do normal cells, areas with lymphoma cells look brighter on the pictures.

Bone marrow biopsy : To check for lymphoma cells in the bone marrow, our doctor will use a thick needle to remove a small sample of bone and bone marrow from our hipbone or another large bone. Local anesthesia can help control pain.

Other staging tests may include biopsies of lymph nodes or other tissue.

Stages of Hodgkin's lymphoma: The stage of Hodgkin lymphoma depends mainly on:

- The number and location of lymph nodes that have Hodgkin lymphoma cells
- Whether the disease has spread to the spleen, liver, bone marrow, lung, or bone

Stages of Hodgkin lymphoma : Oncologists describe the stages of Hodgkin lymphoma using the Roman numerals I, II, III, and IV. Stage I is early-stage cancer, and Stage IV is advanced cancer, such as Hodgkin lymphoma that has spread to the liver.

Stage I : Lymphoma cells are in one lymph node group (such as the lymph nodes in the neck or armpit). Very rarely, Hodgkin lymphoma may start somewhere in the body other than a lymph node and lymphoma cells are found in only that one part.

Stage II : Lymphoma cells are in at least two lymph node groups, but both groups are on the same side of the diaphragm or lymphoma cells are in one part of a tissue or an organ and the lymph nodes near that organ on the same side of the diaphragm. Lymphoma cells may be in other lymph node groups on the same side of the diaphragm.

Stage III : Lymphoma cells are in lymph nodes on both sides of the diaphragm. Lymphoma cells may also be found in one part of a tissue or an organ near these lymph node groups or in the spleen.

Stage IV : Lymphoma cells are found in several parts of at least one organ or tissue. Or, lymphoma cells are in an organ (such as the liver, lung, or bone) and in lymph nodes on the other side of the diaphragm.

In addition to using the Roman numerals I, II, III, and IV, doctors describe the stages of Hodgkin lymphoma with the letters A, B, E, and S. "A" without symptoms such as weight loss, drenching night sweats, or fevers where as "B" with symptoms such as weight loss, drenching night sweats, or fevers. Doctors may describe the stage with an E, S, or both letters. "E" (outside the lymph system), Lymphoma is found in tissues outside the lymph system, such as in liver or lung tissue. Other less common places to find lymphoma include the bone, bone marrow, skin, digestive tract, kidneys, ovaries, or testicles. For example, the stage may be IIE. S (in the spleen) means lymphoma is found in the spleen. For example, the stage may be IIIS.

Symptoms of Hodgkin's lymphoma : People with Hodgkin's disease may experience any of the following signs and symptoms:

- The most common symptom of Hodgkin's is the painless enlargement of one or more

lymph nodes, or lymphadenopathy. The nodes may also feel rubbery and swollen when examined. The nodes of the neck and shoulders (cervical and supraclavicular) are most frequently involved (80–90% of the time, on average). The lymph nodes of the chest are often affected, and these may be noticed on a chest radiograph.

- Persistent fatigue
- Fever and chills
- Night sweats
- Unexplained weight loss — as much as 10 percent or more of your body weight
- Loss of appetite
- Itching
- Increased sensitivity to the effects of alcohol or pain in your lymph nodes after drinking alcohol.
- **Splenomegaly** : enlargement of the spleen occurs in about 30% of people with Hodgkin's lymphoma. The enlargement, however, is seldom massive and the size of the spleen may fluctuate during the course of treatment.
- **Hepatomegaly** : enlargement of the liver, due to liver involvement, is present in about 5% of cases.
- **Hepatosplenomegaly** : the enlargement of both the liver and spleen caused by the same disease.
- **Back pain** : nonspecific back pain (pain that cannot be localized or its cause determined by examination or scanning techniques) has been reported in some cases of Hodgkin's lymphoma. The lower back is most often affected.
- Red-coloured patches on the skin, easy bleeding and petechiae due to low platelet count (as a result of bone marrow infiltration, increased trapping in the spleen etc.—i.e. decreased production, increased removal)
- **Systemic symptoms** : about one-third of patients with Hodgkin's disease may also present with systemic symptoms, including low-grade fever; night sweats; unexplained weight loss of at least 10% of the patient's total body mass in six months or less, itchy skin (pruritus) due to increased levels of eosinophils in the bloodstream; or fatigue (lassitude). Systemic symptoms such as fever, night sweats, and weight loss are known as B symptoms; thus, presence of fever, weight loss, and night sweats indicate that the patient's stage is, for example, 2B instead of 2A.
- **Cyclical fever** : patients may also present with a cyclical high-grade fever known as the Pel-Ebstein fever, or more simply "P-E fever". However, there is debate as to whether the P-E fever truly exists.
- Nephrotic syndrome can occur in individuals with Hodgkin's lymphoma and is most commonly caused by minimal change disease.

Hodgkin lymphoma in the bone marrow : About 1 in 20 people (5%) have Hodgkin lymphoma in their bone marrow when they are diagnosed. It can cause the following effects-

- Breathlessness and tiredness (because of anaemia from a low red blood cell count)
- Increased risk of infections (because of a low white cell count)
- Bleeding problems such as nosebleeds, very heavy periods, or a rash of tiny blood spots under the skin (because of a low platelet count)

Other possible symptoms: Enlarged lymph nodes can

- Press on nerves and cause pain
- Cause swelling in arms or legs by blocking normal tissue fluid circulation
- Yellowing of the skin and eyes (jaundice) by blocking the flow of bile from the liver.

Diagnosis of Hodgkin's lymphoma : Hodgkin's lymphoma must be distinguished from non-cancerous causes of lymph node swelling (such as various infections) and from other types of cancer. Definitive diagnosis is by lymph node biopsy (usually excisional biopsy with microscopic examination). Blood tests are also performed to assess function of major organs and to assess safety for chemotherapy. Positron emission tomography (PET) is used to detect small deposits that do not show on CT scanning. PET scans are also useful in functional imaging (by using a radiolabeled glucose to image tissues of high metabolism). In some cases a Gallium Scan may be used instead of a PET scan.

Medical history and physical exam : If symptoms suggest child might have Hodgkin disease, doctor will want to get a thorough medical history, including information about symptoms, possible risk factors, family history, and other medical conditions. Next, the doctor will do a physical exam, paying special attention to the lymph nodes and other areas of the body that might be affected, including the spleen and liver. Because infections are the most common cause of enlarged lymph nodes, especially in children, the doctor will look for an infection in the part of the body near any swollen lymph nodes. The doctor also might order blood tests to look for signs of infection or other problems. If the doctor suspects that Hodgkin disease might be causing the symptoms, he or she will recommend a biopsy of the area.

Biopsies : Many of the symptoms of Hodgkin disease are actually more likely to be caused by something else. For example, enlarged lymph nodes are more often caused by infections than by Hodgkin disease. Because of this, doctors often wait a few weeks to see if they shrink on their own as the infection goes away. Antibiotics may also be prescribed to see if they cause the nodes to shrink. If the nodes don't shrink or continue to grow, a lymph node (or a small piece of a node) is removed to be looked at under the microscope and for other lab tests. This procedure, called a biopsy, is needed to be sure of the diagnosis. If it is Hodgkin disease, the biopsy can also tell what type it is. There are different types of biopsies such as-

Excisional or incisional biopsy : This is the preferred and most common type of biopsy for an enlarged lymph node. The doctor cuts through the skin to remove the lymph node. If

the doctor removes the entire lymph node, it is called an excisional biopsy. If a small part of a larger tumor or node is removed, it is called an incisional biopsy. If the node is just under the skin, this is a fairly simple operation that can sometimes be done with numbing medicine (local anesthesia). But if the node is inside the chest or abdomen, the patient is sedated or given general anesthesia (where he or she is in a deep sleep). This type of biopsy almost always provides enough of a tissue sample to make a diagnosis of Hodgkin disease and to tell the exact type.

Fine needle aspiration (FNA) or core needle biopsy : In an FNA biopsy, the doctor uses a very thin, hollow needle attached to a syringe to withdraw (aspirate) a small amount of fluid and tiny bits of tissue from a lymph node or an organ in the body. For a core needle biopsy, the doctor uses a larger needle to remove a slightly larger piece of tissue. If the enlarged node is just under the skin, the doctor can aim the needle while feeling the node. If a tumor is deep inside the body, the doctor can guide the needle using a computed tomography (CT) scan or ultrasound. A needle biopsy does not require an incision, but in many cases it might not remove enough of a sample to diagnose Hodgkin disease (or to determine which type it is). Most doctors do not use needle biopsies (especially FNA biopsies) to diagnose Hodgkin disease. But if the doctor suspects that our lymph node swelling is caused by an infection or by the spread of cancer from another organ (such as the breast, lungs, or thyroid), a needle biopsy might be the first type of biopsy done. An excisional biopsy may still be needed to diagnose Hodgkin disease, even after a needle biopsy has been done. If Hodgkin disease has already been diagnosed, needle biopsies are sometimes used to check areas in other parts of the body that might be Hodgkin disease spreading or coming back after treatment.

Bone marrow aspiration and biopsy : These tests are not used to diagnose Hodgkin disease, but they may be done after the diagnosis is made to see if Hodgkin disease is in the bone marrow.

Lab tests of biopsy samples : All biopsy samples are looked at under a microscope by a pathologist (a doctor specially trained to recognize cancer cells), who looks at the size and shape of the cells and determines if any of them are Reed-Sternberg cells. The pathologist also looks at how the cells are arranged, which could point to the type of Hodgkin disease. Because diagnosing Hodgkin disease can be tricky, it helps if the pathologist specializes in diseases of the blood. Sometimes the first biopsy does not give a definite answer and more biopsies are needed.

Immunohistochemistry : In this test, a part of the biopsy sample is treated with special antibodies (man-made versions of immune system proteins) that will attach only to certain molecules on the surface of cells. These antibodies cause color changes that can be seen under a microscope. This test can show certain proteins, such as CD15 and CD30, on the surface of the Reed-Sternberg cells. These are typically found in classic Hodgkin disease. Tests for other proteins may point to nodular lymphocyte predominant Hodgkin disease, to non-Hodgkin lymphoma rather than Hodgkin disease, or to other diseases entirely.

NON-HODGKIN'S LYMPHOMA

Non-Hodgkin lymphoma (NHL) is cancer of the lymph tissue. Lymph tissue is found in the lymph nodes, spleen, and other organs of the immune system. White blood cells called lymphocytes are found in lymph tissue. Most lymphomas start in a type of white blood cell called B lymphocyte, or B cell.

History of Non-Hodgkin Lymphoma : Hodgkin's Lymphoma (HL, Hodgkin's disease), described by Thomas Hodgkin in 1832, was the first form of lymphoma described and defined. Other forms were later described and there was a need to classify them. Because Hodgkin's lymphoma was much more radiation-sensitive than other forms, its diagnosis was important for oncologists and their patients. The first classification of Hodgkin's Lymphoma was proposed by Robert J. Lukes in 1963. While consensus was rapidly reached on the classification of Hodgkin's lymphoma, there remained a large group of very different diseases requiring further classification. The Rappaport classification, proposed by Henry Rappaport in 1956 and 1966, became the first widely accepted classification of lymphomas other than Hodgkin's. Following its publication in 1982, the Working Formulation became the standard classification for this group of disease. It introduced the term non-Hodgkin's Lymphoma (NHL) and defined three grades of lymphoma. However, NHL consists of 16 different conditions that have little in common with each other. They are grouped by their aggressiveness. Less aggressive non-Hodgkin lymphomas are compatible with a long survival while more aggressive non-Hodgkin lymphomas can be rapidly fatal without treatment. Without further narrowing, the label is of limited usefulness for patients or doctors. The most recent lymphoma classifications, the 1994 Revised European-American Lymphoma (REAL) classification and the 2001 WHO classification, abandoned the HL vs. NHL grouping. Instead, 43 different forms of lymphoma are listed and discussed separately. Although Hodgkin's lymphoma is recognised as being a tumour of lymphocytes of mature B cell lineage, it is still considered separately within the WHO classification.

Risk factors for Non-Hodgkin lymphoma : The International Agency for Research on Cancer (IARC) evaluates evidence on the carcinogenic risk to humans of a number of exposures including tobacco, alcohol, infections, radiation, occupational exposures, and medications.⁵¹ The World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) evaluates evidence for other exposures including diet, overweight and obesity, and physical exercise. NHLs may result from chromosomal translocations, infections, environmental factors, immunodeficiency states, and chronic inflammation.

Chromosomal translocations : Chromosomal translocations and molecular rearrangements play an important role in the pathogenesis of many lymphomas and correlate with histology and immunophenotype. The t(14;18)(q32;q21) translocation is the most common chromosomal abnormality associated with NHL. This translocation occurs in 85%

of follicular lymphomas and 28% of higher-grade NHLs. This translocation results in the juxtaposition of the bcl-2 apoptotic inhibitor oncogene at chromosome band 18q21 to the heavy chain region of the immunoglobulin (Ig) locus within chromosome band 14q32. The t(11;14)(q13;q32) translocation has a diagnostic nonrandom association with mantle cell lymphoma. This translocation results in the overexpression of bcl-1 (cyclin D1/PRAD 1), a cell-cycle regulator on chromosome band 11q13. The 8q24 translocations lead to c-myc dysregulation. This is frequently observed in high-grade small noncleaved lymphomas (Burkitt and non-Burkitt types), including those associated with HIV infection. The t(2;5)(p23;q35) translocation occurs between the nucleophosmin (NPM) gene and the anaplastic lymphoma kinase (ALK1) gene. It results in the expression of an aberrant fusion protein found in a majority of anaplastic large cell lymphomas. Two chromosomal translocations, t(11;18)(q21;q21) and t(1;14)(p22;132), are associated with mucosa-associated lymphoid tissue (MALT) lymphomas. The more common (ie, t[11;18](q21;q21)) translocates the apoptosis inhibitor AP12 gene with the MALT1 gene, resulting in the expression of an aberrant fusion protein. The other translocation, t(1;14)(p22;132), involves the translocation of the bcl-10 gene to the immunoglobulin gene enhancer region.

Infections: NHL risk is associated with a number of infections, because immune deregulation plays a pivotal role in lymphomagenesis (the development of lymphoma). Some viruses are implicated in the pathogenesis of NHL, probably because of their ability to induce chronic antigenic stimulation and cytokine deregulation, which leads to uncontrolled B or T-cell stimulation, proliferation, and lymphomagenesis. Epstein-Barr virus (EBV) is a DNA virus that is associated with Burkitt lymphoma (especially the endemic form in Africa), Hodgkin disease, lymphomas in immunocompromised patients (eg, from HIV infection, organ transplantation), and sinonasal lymphoma.

Human immunodeficiency virus (HIV)/AIDS : Human immunodeficiency virus (HIV) type I is classified by IARC as a cause of NHL, estimated 0.6% of NHL cases in the UK are linked to HIV. NHL risk is around 11 times higher in people with HIV, compared with the general population. NHL risk in HIV-positive people increases with HIV severity. HIV-associated NHL risk has decreased markedly since highly active antiretroviral therapy (HAART) was introduced to treat HIV in 1996 because HAART improves immune system functioning.

Human T-lymphotropic virus type 1 (HTLV-1) : Human T-cell leukemia virus type 1 (HTLV-1) causes a latent infection via reverse transcription in activated T-helper cells. This virus is endemic in certain areas of Japan and the Caribbean islands, and approximately 5% of carriers develop adult T-cell leukemia or lymphoma.

Hepatitis C viruses (HCV) : Hepatitis C virus (HCV) is associated with the development of clonal B-cell expansions and certain subtypes of NHL (ie, lymphoplasmacytic lymphoma, Waldenström macroglobulinemia), especially in the setting of essential (type II) mixed cryoglobulinemia.

Kaposi sarcoma herpes virus (KSHV) : Kaposi sarcoma associated herpesvirus (KSHV) is associated with body cavity–based lymphomas in patients with HIV infection and in patients with multicentric Castlemans disease.

Helicobacter pylori (H. Pylori) : Helicobacter pylori infection is associated with the development of primary gastrointestinal (GI) lymphomas, particularly gastric mucosa-associated lymphoid tissue (MALT) lymphomas.

Epstein-Barr virus (EBV) : NHL risk is 26% higher in people with previous infectious mononucleosis (which is caused by EBV), NHL risk is higher in EBV-positive people, a meta-analysis showed.

Immune system : Higher NHL risk in people with autoimmune conditions and organ transplant recipients may relate to use of immunosuppressant medication, as well as to underlying medical condition.

Autoimmune conditions : NHL risk is 14-19 times higher in people with primary Sjogrens syndrome; 7 times higher in people with systemic lupus erythematosus (SLE); 2-4 times higher in people with rheumatoid arthritis; 2-6 times higher in people with coeliac disease (enteropathy-associated T-cell lymphomas – EATCL/EATL), and 2.7 times higher in people with systemic sclerosis, compared with the general population, meta-analyses. The NHL subtypes most often associated with autoimmunity are diffuse large B-cell lymphoma (DLBCL) and marginal zone lymphomas (MZL). Immune deregulation underpins both autoimmune disease and lymphoma development. NHL risk (B-cell NHL subtypes) in inflammatory rheumatic disease patients may be higher in those receiving antitumor necrosis factor alpha (anti-TNF α) treatment.

Organ transplant : NHL risk is around 8 times higher in organ transplant recipients receiving immunosuppressant medication, cohort and case-control studies have shown; these patients usually develop DLBCL.

Family history and genetic conditions

Family history : NHL risk is higher in people with a first-degree relative (parent, sibling, and child) with NHL, a pooled analysis and cohort studies have shown that relatives may develop the same NHL subtype. NHL risk may be higher in people with a first degree relative with other cancer types.

Previous cancer : NHL risk is higher in people with previous cancer of various types. The most consistent links are with previous Hodgkin lymphoma or leukaemia, but risk may also be higher in survivors of melanoma.

Overweight and obesity : DLBCL risk is 13-14% higher in overweight people (by BMI) and 29% higher in obese people, compared with healthy weight people. DLBCL and follicular lymphoma (FL) risk is also higher in those who had higher BMI in young adulthood.

Ionizing radiation : X radiation and gamma radiation are classified by IARC as probable causes of NHL, based on limited evidence.

Occupational exposures : Working in rubber production is a cause of NHL, and benzene, ethylene oxide, Tetrachlorodibenzo-para-dioxin, polychlorophenols or their sodium salts (combined exposures), tetrachloroethylene, and trichloroethylene are probable causes of NHL. NHL risk is higher in people with occupational exposure to trichloroethylene or pesticides.

Reproductive and hormonal factors:

Postmenopausal hormone replacement therapy : NHL risk is 22-26% lower in women who started using hormone replacement therapy (HRT) at age 50+, compared with never-users; NHL risk is reduced in current HRT users but not past users.

Oral contraceptives (OCs) : NHL risk is 46% higher in women who started using oral contraceptives (OCs) aged 22+, compared with never-users, this may be limited to use before the mid-1970s, when hormone doses in OCs were higher than today.

Factors shown to decrease or have no effect on NHL risk :

Decrease risk : NHL risk is 19% lower in people with the highest vegetable intake, versus those with the lowest. Alcohol consumption may reduce risk of NHL, meta- and pooled analyses have shown though there was no dose-response effect, and the association was largely limited to case-control studies, calling into question the mechanism and validity of the association. NHL risk is lower in people with alcohol use disorders, a cohort study showed. NHL risk is not associated with the following factors-

- Tobacco smoking.
- Fruit
- Age at menarche
- Age at menopause
- Physical activity

Types of non-Hodgkin lymphoma : Classifying non-Hodgkin lymphoma can be quite confusing because there are so many types and several different systems have been used. The most recent system is the World Health Organization (WHO) classification. The WHO system groups lymphomas based on how they look under a microscope, the chromosome features of the lymphoma cells, and the presence of certain proteins on the surface of the cells. (Older systems classified lymphomas only by how they looked under a microscope.) The more common types of lymphoma are listed below according to whether they are B-cell or T-cell lymphomas.

B-cell lymphomas : B-cell lymphomas make up most (about 85%) of non-Hodgkin lymphomas in the United States.

Diffuse large B-cell lymphoma : This is the most common type of non-Hodgkin lymphoma in the United States, accounting for about 1 out of every 3 cases. The cells are fairly large when seen using a microscope. Diffuse large B-cell lymphoma (DLBCL) can affect any age group but occurs mostly in older people (the average age is mid-60s). It usually starts as a quickly growing mass in a lymph node deep inside the body, such as in the chest or abdomen, or in a lymph node that you can feel, such as in the neck or armpit. It can also start in other areas such as the intestines, bone, or even the brain or spinal cord. About 1 in 3 of these lymphomas is confined to one part of the body (localized) when it is found. Lymphomas are easier to treat when they are localized than when they have spread to other parts of the body. Genetic tests have shown that there are different subtypes of DLBCL, even though they look the same under the microscope. These subtypes seem to have different outlooks (prognoses) and responses to treatment. DLBCL is a fast growing lymphoma, but it often responds well to treatment. Overall, about 3 out of 4 people will have no signs of disease after the initial treatment, and about half of all people with this lymphoma are cured with therapy.

Primary mediastinal B-cell lymphoma : This is a subtype of DLBCL in which the lymphoma cells are large but there is a lot of fibrosis (scar-like tissue) in the background. It accounts for about 2% of all lymphomas. About 2 out of 3 people with this lymphoma are women. Most are young in their 30s. This lymphoma starts in the mediastinum (the area in the middle of the chest behind the breastbone). It is usually localized when it is found. It can cause trouble breathing because it often presses on the windpipe (trachea) leading into the lungs. It can also block the superior vena cava (the large vein that returns blood to the heart from the arms and head), which can make the arms and face swell. This is a fast-growing lymphoma, but usually responds well to treatment and half of patients can be cured.

Intravascular large B-cell lymphoma : In this rare subtype of DLBCL, the lymphoma cells are only found inside blood vessels, not in the lymph nodes or bone marrow. It is treated like DLBCL.

Follicular lymphoma : About 1 out of 5 lymphomas in the United States is follicular lymphoma. The term follicular means that the cells tend to grow in a circular pattern in lymph nodes. The average age for people with this lymphoma is about 60 and rare in very young people. Most of the time, this lymphoma occurs in many lymph node sites in the body, as well as in the bone marrow. Follicular lymphomas are often slow-growing and respond well to treatment, but they are hard to cure. These lymphomas may not require treatment when they are first diagnosed. Instead, treatment may be delayed until the lymphoma is causing problems. Over time, about 1 in 3 follicular lymphomas turns into a fast-growing diffuse B-cell lymphoma.

Chronic lymphocytic leukemia /small lymphocytic lymphoma : These are closely related diseases. In fact, many doctors consider them different versions of the same disease. The same type of cancer cell (known as a small lymphocyte) is seen in both chronic lymphocytic leukemia (CLL) and small lymphocytic lymphoma (SLL). The only difference is where the

cancer cells are found. In CLL most of the cancer cells are in the blood and bone marrow. In SLL, the cancer cells are mainly in the lymph nodes and spleen. SLL accounts for about 5% to 10% of all lymphomas. Both CLL and SLL are slow-growing diseases, although CLL, which is much more common, tends to grow more slowly. CLL and SLL are treated the same way. They are usually not curable with standard treatments, but depending on the stage and growth rate of the disease, most patients live longer than 10 years. Sometimes, these slow-growing lymphomas turn into a more aggressive type of lymphoma. Only about 5% of lymphomas are this type. The cells are small to medium in size. Men are affected most often. The average age of patients is in the early 60s. When this lymphoma is diagnosed, it is usually widespread in the lymph nodes, bone marrow, and often the spleen. This usually isn't a very fast-growing lymphoma, but it can be challenging to treat. Newer treatments might be more effective than those used in the past, and may offer a better chance for long-term survival for patients now being diagnosed.

Marginal zone B-cell lymphomas : Marginal zone lymphomas account for about 5% to 10% of lymphomas. The cells in these lymphomas look small under the microscope. There are 3 main types of marginal zone lymphomas. Extranodal marginal zone B-cell lymphomas, also known as mucosa-associated lymphoid tissue (MALT) lymphomas: These lymphomas start in places other than the lymph nodes (extranodal) and are the most common type. Most MALT lymphomas start in the stomach and are linked to infection by the bacteria *Helicobacter pylori*, which is also the cause of stomach ulcers. Other possible sites of MALT lymphomas include the lung, skin, thyroid, salivary glands, and tissues surrounding the eye. Usually it is confined to the area where it begins and is not widespread. Many of these other MALT lymphomas have also been linked to infections with bacteria or viruses. The average age of patients with MALT lymphoma is about 60. It is a slow-growing lymphoma and is often curable in its early stages. Doctors often use antibiotics as the first treatment for MALT lymphoma of the stomach, because treating the *Helicobacter pylori* infection often cures the lymphoma.

Nodal marginal zone B-cell lymphoma : This is a rare disease, found mainly in older women. It usually stays in the lymph nodes, although lymphoma cells can also sometimes be found in the bone marrow. This tends to be a slow-growing lymphoma (although not usually as slow as MALT lymphoma), and many patients are cured if they are diagnosed when the disease is in the early stages.

Splenic marginal zone B-cell lymphoma : This is a rare lymphoma. Most often the lymphoma is found only in the spleen and bone marrow. Male patients are often elderly have fatigue and discomfort caused by an enlarged spleen. Because the disease is slow-growing, treatment may not be needed unless the symptoms become troublesome. This type of lymphoma has been linked to infection with the hepatitis C virus.

Burkitt lymphoma : This type makes up about 1% to 2% of all lymphomas. It is named after the doctor who first described this disease in African children and young adults. The

cells are medium sized. Another kind of lymphoma, called Burkitt-like lymphoma, has slightly larger cells. Because this second kind of lymphoma is hard to tell apart from Burkitt lymphoma, the WHO classification combines them. This is a very fast-growing lymphoma. In the African (or endemic) variety, it often starts as tumors of the jaws or other facial bones. This type is linked to infection with the Epstein-Barr virus (which can also cause infectious mononucleosis or “mono”). The endemic type of Burkitt lymphoma is rare in the United States. In the types seen more often in the United States, the lymphoma usually starts in the abdomen, where it forms a large tumor mass. It can also start in the ovaries, testicles, or other organs, and can spread to the brain and spinal fluid. The type seen in the United States is usually not linked to Epstein-Barr viral infection. Close to 90% of patients are male, and the average age in the US is about 30. Although this is a fast-growing lymphoma, more than half of patients can be cured by intensive chemotherapy.

Lymphoplasmacytic lymphoma (Waldenstrom macroglobulinemia) : Waldenstrom macroglobulinemia (WM) or lymphoplasmacytic lymphoma is a type of non-Hodgkin lymphoma (NHL) in which the cancer cells make large amounts of an abnormal protein (called a macroglobulin). This type is not common, accounting for 1% to 2% of lymphomas. The lymphoma cells are small and found mainly in the bone marrow, lymph nodes, and spleen. WM is a cancer that starts in B cells. The cancer cells in people with WM are similar to those of 2 other types of cancer: multiple myeloma and non-Hodgkin lymphoma. Multiple myeloma is considered a cancer of plasma cells and non-Hodgkin lymphoma is a cancer of lymphocytes. WM cells have features of both plasma cells and lymphocytes and are called lymphoplasmacytoid. WM cells make large amounts of a certain type of antibody (immunoglobulin M, or IgM), which is known as a macroglobulin. Each protein made by the WM cells is the same, so it is called a monoclonal protein, or just an M protein. The buildup of this M protein in the body can lead to many of the symptoms of WM, including excess bleeding, problems with vision, and nervous system problems. The WM cells grow mainly in the bone marrow, where they can crowd out the normal cells that make the different types of blood cells. This can lead to low levels of red blood cells (called anemia), which can make people feel tired and weak. It can also cause low numbers of white blood cells, which makes it hard for the body to fight infection. The numbers of platelets in the blood can also drop, leading to increased bleeding and bruising.

Hairy cell leukemia : Despite the name, this is sometimes considered to be a type of lymphoma. Hairy cell leukemia (HCL) is rare about 700 people in the United States are diagnosed with it each year. The cells are small B lymphocytes with projections coming off them that give them a “hairy” appearance. They are typically found in the bone marrow and spleen in the blood. Men are more likely to get HCL than women, and the average age is around 50. Hairy cell leukemia is slow-growing, and some patients may never need treatment. An enlarging spleen or dropping blood cell counts (due to cancer cells invading the bone marrow) are the usual reasons to begin treatment. If treatment is needed, it's usually very

effective.

Primary central nervous system (CNS) lymphoma : This lymphoma usually involves the brain (called primary brain lymphoma), but it may also be found in the spinal cord and in tissues around the spinal cord and the eye. Over time, it tends to become widespread in the central nervous system. Primary CNS lymphoma is rare overall, but it's more common in people with immune system problems, such as those infected with HIV, the virus that causes AIDS. Most people develop headaches and confusion. They can also have vision problems; weakness or altered sensation in the face, arms, or legs; and in some cases, seizures. The outlook for people with this condition has always been thought to be fairly poor, but some people can live at least 5 years with today's treatments.

T-cell lymphomas : T-cell lymphomas make up less than 15% of non-Hodgkin lymphomas in the United States. There are many types of T-cell lymphoma, but they are all fairly rare.

Precursor T-lymphoblastic lymphoma/leukemia : This disease accounts for about 1% of all lymphomas. It can be considered either a lymphoma or leukemia, depending on how much of the bone marrow is involved (leukemias have more bone marrow involvement). The cancer cells are small-to-medium sized, immature T-cells. This lymphoma often starts in the thymus where many T cells are made. This lymphoma can develop into a large tumor in the mediastinum (the area in the middle of the chest and behind the breast bone). If the tumor presses on the windpipe (trachea) that carries air into the lungs, it can cause trouble breathing. The tumor can also press on or even block the superior vena cava (the large vein that returns blood to the heart from the arms and head), which can make the arms and face swell. Patients are most often young adults, with males being affected more often than females. This lymphoma is fast-growing, but if it hasn't spread to the bone marrow when it is first diagnosed, the chance of cure with chemotherapy is quite good.

Peripheral T-cell lymphomas : These types of lymphomas develop from more mature forms of T cells. They are rare, accounting for only a small portion of all lymphomas.

Cutaneous T-cell lymphomas (mycosis fungoides, Sezary syndrome, and others) : These lymphomas start in the skin. Skin lymphomas account for about 5% of all lymphomas.

Angioimmunoblastic T-cell lymphoma : This lymphoma accounts for about 3% of all lymphomas. It tends to occur in the lymph nodes and may affect the spleen or liver. Patients usually have fever, weight loss, and skin rashes and often develop infections. This lymphoma often progresses quickly. Treatment is often effective at first, but the lymphoma tends to come back.

Extranodal natural killer/T-cell lymphoma, nasal type : This rare type often involves the upper airway passages, such as the nose and upper throat, but it can also invade the skin and digestive tract. It is much more common in parts of Asia and South America. Cells of this lymphoma are similar in some ways to normal natural killer (NK) cells. NK cells are a kind of lymphocyte that can respond to infections more quickly than T-cells and B-cells.

Enteropathy type intestinal T-cell lymphoma : This lymphoma occurs in people with extreme sensitivity to gluten, the main protein in wheat flour. People with this disease, called gluten-sensitive enteropathy (celiac sprue or sprue), can develop lymphoma in the walls of the intestines. This is rare in people found to have sprue in childhood, and is more often found in adults either recently diagnosed with sprue or who didn't realize that they had sprue until part of the intestine was removed to treat the lymphoma. Many experts believe that these patients had sprue for a long time without knowing it. It is thought that if sprue is found early, following a gluten-free diet may help prevent the lymphoma from developing. People who do not tolerate gluten, but don't have sprue, do not seem to have an increased risk of this type of lymphoma.

Anaplastic large cell lymphoma (ALCL) : About 2% of lymphomas are of this type. It is more common in young people (including children), but it does occur in people in their 50s and 60s. It usually starts in lymph nodes and can also spread to skin. This type of lymphoma tends to be fast-growing, but many people with this lymphoma are cured with aggressive chemotherapy. There are 2 main forms of ALCL, systemic ALCL and primary cutaneous ALCL, which only affects the skin. Systemic ALCL is divided into 2 types based on whether a gene change is present in the lymphoma cells that causes them to make a lot of protein called anaplastic lymphoma kinase or ALK1. ALK-positive ALCL tends to have a better prognosis (outlook) than the ALK-negative type.

Peripheral T-cell lymphoma, unspecified : This name is given to T-cell lymphomas that don't readily fit into any of the groups above. They make up about half of all T-cell lymphomas. The tumor cells can be small or large. Most people diagnosed with this disease are in their 60s. As a group, these lymphomas tend to be widespread and grow quickly. Some cases respond well to chemotherapy, but long-term survival is not common.

Signs and symptoms of non-Hodgkin lymphoma : Non-Hodgkin lymphoma can cause many different signs and symptoms, depending on where it is in the body. In some cases it might not cause any symptoms until it grows quite large. Common symptoms include:

- Enlarged lymph nodes
- Swollen abdomen (belly)
- Feeling full after only a small amount of food
- Chest pain or pressure
- Shortness of breath or cough
- Fever
- Weight loss
- Night sweats
- Fatigue (extreme tiredness)

Swollen lymph nodes : Non-Hodgkin lymphoma can cause lymph nodes to become enlarged. When this occurs in lymph nodes close to the surface of the body (such as on the sides of the neck, in the groin or underarm areas, or above the collar bone), they may be seen or felt as lumps under the skin. These are often found by the patient, a family member, or a health care professional. Although enlarged lymph nodes are a common symptom of lymphoma, they are much more often caused by infections.

Lymphoma in the abdomen : Lymphomas in the abdomen may cause it to become swollen and tender. This may be because of lymph nodes in the abdomen enlarging, but it can also be caused by the build-up of large amounts of fluid. Lymphoma can cause the spleen to become enlarged and press on the stomach. This can cause a person to feel full after eating only a small amount of food. When lymphoma is in the intestines or causes swelling near the intestines, bowel movements may be blocked, which may lead to abdominal pain, nausea, or vomiting. Lymphoma in the intestines can also cause holes to develop in the intestine wall (called perforations). This allows the contents of the intestines to leak out into the abdominal cavity, leading to serious infection and severe pain with nausea and vomiting. Lymphomas of the stomach often cause stomach pain, nausea, and reduced appetite.

Lymphoma in the chest : When lymphoma starts in the thymus or lymph nodes in the chest, it may press on the nearby trachea (windpipe), which can cause coughing or trouble breathing. Lymphomas in this area can also cause a feeling of chest pain or pressure. The superior vena cava (SVC) is the large vein that carries blood from the head and arms back to the heart. It passes near the thymus and lymph nodes inside the chest. Lymphomas in this area may push on the SVC, which can cause the blood to back up in the veins. This can lead to swelling (and sometimes a bluish-red color) in the head, arms, and upper chest. It can also cause trouble breathing and a change in consciousness if it affects the brain. This condition, known as SVC syndrome, can be life-threatening and must be treated right away.

Lymphoma affecting the brain : Lymphomas of the brain, called primary brain lymphomas, can cause headache, trouble thinking, and weakness in certain parts of the body, personality changes, and sometimes seizures. Other types of lymphoma can spread to the area around the brain and spinal cord. This can cause problems such as double vision, facial numbness, and trouble speaking.

Lymphoma in the skin : Lymphomas of the skin may be seen or felt. They often appear as extremely itchy, red or purple lumps or nodules under the skin.

General symptoms : Along with causing symptoms and signs in the part of the body where it starts, non-Hodgkin lymphoma can also cause general symptoms, such as:

- Unexplained weight loss
- Fever
- Drenching night sweats (enough to soak clothing and sheets)

When talking about lymphoma, doctors call these B symptoms. The presence of

B symptoms is most common in more rapidly growing lymphomas. These symptoms are important not only in helping diagnose non-Hodgkin lymphoma, but also in determining the stage and prognosis. Other symptoms can be caused by low blood counts. Blood counts can become low when lymphoma spreads to the bone marrow and crowds out the normal, healthy cells that make new blood cells. Anemia can also occur if the lymphoma cells cause the body to destroy red blood cells (this is called hemolytic anemia). This can lead to problems like:

- Severe or frequent infections (from low white blood cell counts)
- Easy bruising or bleeding (from low blood platelet counts)
- Fatigue (anemia from low red blood cell counts)

Medical history and physical exam : If the symptoms suggest non-Hodgkin lymphoma, doctor will want to get a thorough medical history, including information about symptoms, possible risk factors, family history, and other medical conditions. Next, the doctor will pay special attention to the lymph nodes and other areas of the body that might be involved, including the spleen and liver. Because infections are the most common cause of enlarged lymph nodes, the doctor will look for an infection in the part of the body near the swollen lymph nodes. If the doctor suspects that non-Hodgkin lymphoma might be causing the symptoms, he or she will recommend a biopsy of the area.

Biopsy : generally symptoms of non-Hodgkin lymphoma are not specific enough to say for certain if they are being caused by cancer. Most of these symptoms can also be caused by non-cancerous problems, like infections, or by other kinds of cancers. For example, enlarged lymph nodes are more often caused by infections than by non-Hodgkin lymphoma. Because of this, doctors often prescribe antibiotics and wait a few weeks to see if the nodes shrink. If the nodes stay the same or continue to grow, the doctor might then advise a biopsy. Either a small piece of a node or, more commonly, the entire node is removed for viewing under the microscope and for other lab tests. A biopsy might be needed right away if the size, texture, or location of the node or the presence of other symptoms strongly suggests cancer. But a delay in diagnosis of a few weeks is not likely to be harmful unless it's a very rapidly growing lymphoma.

Types of biopsies used to diagnose non-Hodgkin lymphoma : A biopsy is the only way to diagnose non-Hodgkin lymphoma. There are several types of biopsies. Doctors choose which one to use based on the unique aspects of each person's situation.

Excisional or incisional biopsy : This is the most common type of biopsy if lymphoma is suspected. In this procedure, a surgeon cuts through the skin to remove either the entire node (excisional biopsy) or a small part of a large tumor (incisional biopsy). If the node is near the skin surface, this is a simple operation that can often be done with local anesthesia (numbing medicine). But if the node is inside the chest or abdomen, the patient is sedated or given general anesthesia. This method almost always provides enough of a sample to diagnose the exact type of non-Hodgkin lymphoma. It is the preferred type of biopsy, if it can be done without too much discomfort to the patient.

Fine needle aspiration (FNA) or core needle biopsy : In an FNA biopsy, the doctor uses a very thin, hollow needle attached to a syringe to withdraw (aspirate) a small amount of tissue from an enlarged lymph node or a tumor mass. For a core needle biopsy, the doctor uses a larger needle to remove a slightly larger piece of tissue. For an enlarged node near the surface of the body, the doctor can aim the needle while feeling the node. If the tumor is deep inside the body, the doctor can guide the needle using a computed tomography (CT) scan or ultrasound. A needle biopsy does not require surgery, but in some cases it might not remove enough of a sample to make a definite diagnosis. Most doctors do not use needle biopsies to diagnose lymphoma. But if the doctor suspects that lymph node swelling is caused by an infection or by the spread of cancer from another organ (such as the breast, lungs, or thyroid), a needle biopsy may be the first type of biopsy done. An excisional biopsy might still be needed to diagnose and classify lymphoma, even after a needle biopsy has been done. Once lymphoma has been diagnosed, needle biopsies are sometimes used to check areas in other parts of the body that might be lymphoma spreading or coming back after treatment.

Other types of biopsies : These procedures are not normally done to diagnose lymphoma, but they might be done to help determine the stage (extent) of a lymphoma that has already been diagnosed.

Bone marrow aspiration and biopsy : These procedures are often done after lymphoma has been diagnosed to help determine if it has reached the bone marrow. The two tests are often done at the same time. The samples are usually taken from the back of the pelvic (hip) bone, although in some cases they may be taken from the sternum (breast bone) or other bones. After cleaning the skin over the hip, the doctor numbs the area and the surface of the bone with local anesthetic, which can cause a brief stinging or burning sensation. A thin, hollow needle is then inserted into the bone and a syringe is used to suck out a small amount of liquid bone marrow (about 1 teaspoon). Even with the anesthetic, most patients still have some brief pain when the marrow is removed. A bone marrow biopsy is usually done just after the aspiration. A small piece of bone and marrow is removed with a slightly larger needle that is twisted as it is pushed into the bone. The biopsy might also cause some brief pain. Once the biopsy is done, pressure will be applied to the site to help stop any bleeding.

Lumbar puncture (spinal tap) : This test looks for lymphoma cells in the cerebrospinal fluid (CSF), which is the liquid that bathes the brain and spinal cord. For this test, the patient may lie on their side or sit up. The doctor first numbs an area in the lower part of the back over the spine. A small, hollow needle is then placed between the bones of the spine to withdraw some of the fluid. Most people with lymphoma will not need this test. But doctors may order it for certain types of lymphoma or if a person has symptoms that suggest the lymphoma may have reached the brain.

Pleural or peritoneal fluid sampling : Spread of lymphoma to the chest or abdomen can cause fluid to build up. Pleural fluid (inside the chest) or peritoneal fluid (inside the abdomen) can be removed by placing a hollow needle through the skin into the chest or

abdomen. The doctor uses a local anesthetic to numb the skin before inserting the needle. The fluid is then withdrawn and looked at under the microscope to check for lymphoma cells. When this procedure is used to remove fluid from the area around the lung, it is called a thoracentesis. When it is used to collect fluid from inside the abdomen, it's known as a paracentesis. All biopsy samples and fluids are looked at under a microscope by a pathologist (a doctor with special training in recognizing cancer cells), who studies the size and shape of the cells and how they are arranged. This may reveal not only if the person has a lymphoma, but also what type of lymphoma it is. Because diagnosing lymphoma can be tricky, it helps if the pathologist specializes in diseases of the blood. Pathologists can sometimes tell which kind of lymphoma a patient has by looking at the cells, but usually other types of tests are needed to confirm the diagnosis.

Immunohistochemistry : In this test, a part of the biopsy sample is treated with special antibodies (man-made versions of immune system proteins) that attach only to specific molecules on the cell surface. These antibodies cause color changes, which can be seen under a microscope. This test may be helpful in distinguishing different types of lymphoma from one another and from other diseases.

Flow cytometry : Like immunohistochemistry, this test looks for certain substances on the outside surface of cells that help identify what types of cells they are. But this test can look at many more cells than immunohistochemistry. For this test, a sample of cells is treated with special antibodies that stick to the cells only if certain substances are present on their surfaces. The cells are then passed in front of a laser beam. If the cells now have antibodies attached to them, the laser will cause them to give off light, which can be measured and analyzed by a computer. Groups of cells can be separated and counted by these methods. This is the most commonly used test for immunophenotyping (classifying lymphoma cells according to the substances [antigens] on their surfaces). Different types of lymphocytes have different antigens on their surface. These antigens may also change as each cell matures. Flow cytometry can help determine whether the lymph node is swollen because of lymphoma, some other cancer, or a non-cancerous disease. It has also become very useful in helping doctors determine the exact type of lymphoma so that they can select the best treatment.

Cytogenetics : This technique allows doctors to evaluate the chromosomes (long strands of DNA) in the lymphoma cells. The cells are looked at under a microscope to see if the chromosomes have any translocations (where part of one chromosome has broken off and is now attached to another chromosome), as happens in certain types of lymphoma. Some lymphoma cells may have too many chromosomes, too few chromosomes, or other chromosome abnormalities. These changes can help identify the type of lymphoma. Cytogenetic testing usually takes about 2 to 3 weeks because the lymphoma cells must grow in lab dishes for a couple of weeks before their chromosomes are ready to be viewed under the microscope.

Molecular genetic tests : These tests look more closely at lymphoma cell DNA. They

can detect most changes that are visible under a microscope in cytogenetic tests, as well as others that can't be seen.

Fluorescent in situ hybridization (FISH) : FISH uses special fluorescent dyes that only attach to specific genes or parts of chromosomes. FISH can find most chromosome changes (such as translocations) that can be seen under a microscope in standard cytogenetic tests, as well as some gene changes too small to be seen with usual cytogenetic testing. FISH can be used to look for specific genes or changes in chromosomes. It can be used on regular blood or bone marrow samples. It is very accurate and can usually provide results within a couple of days, which is why this test is now used in many medical centers.

Polymerase chain reaction (PCR) : PCR is a very sensitive DNA test that can find gene changes and certain chromosome changes too small to be seen under a microscope, even if very few lymphoma cells are present in a sample. .

Blood tests : Blood tests measure the amounts of certain types of cells and chemicals in the blood. They are not used to diagnose lymphoma, but they can sometimes help determine how advanced the lymphoma is. Patients with known or suspected lymphoma will have a complete blood count (CBC) checked. This test measures the different cells in the blood, such as the red blood cells, the white blood cells, and the platelets. In patients already known to have lymphoma, low blood cell counts can mean that the lymphoma is growing in the bone marrow and affecting new blood cell formation. Many patients will also have blood chemistry tests run, to look at kidney and liver function. If lymphoma has been diagnosed, another blood test called lactate dehydrogenase (LDH) may be checked. LDH levels are often increased in patients with lymphomas. For some types of lymphoma or if certain treatments may be used, doctor may also advise other blood tests to see if have been infected with certain viruses, such as the hepatitis B virus (HBV), hepatitis C virus (HCV), or human immunodeficiency virus (HIV). Infections with these viruses may affect treatment.

Imaging tests : Imaging tests use x-rays, sound waves, magnetic fields, or radioactive particles to produce pictures of the inside of the body. In someone with known or suspected lymphoma, these tests might be done to look more closely at an abnormal area that might contain lymphoma.

Chest x-ray : X-rays of the chest may be done to look for enlarged lymph nodes in this area.

Computed tomography (CT) scan : The CT scan is an x-ray test that produces detailed, cross-sectional images of body. Instead of taking one picture, like a regular x-ray, a CT scanner takes many pictures. A computer then combines these pictures into an image of a slice of body. Unlike a regular x-ray, CT scans can show the detail in soft tissues (such as internal organs). This scan can help tell if any lymph nodes or organs in body are enlarged. CT scans are useful for looking for lymphoma in the abdomen, pelvis, chest, head, and neck.

Before the test, may be asked to drink a contrast solution and/or get an intravenous (IV) injection of a contrast dye that helps better outline abnormal areas in the body. The injection

can cause some flushing (a feeling of warmth, especially in the face). Some people are allergic and get hives or, rarely, more serious reactions like trouble breathing and low blood pressure. CT scans take longer than regular x-rays. During the test, the table slides in and out of the scanner, a ring-shaped machine that completely surrounds the table. In some cases, CT can be used to guide a biopsy needle into a suspicious area. For this procedure, called a CT-guided needle biopsy, remain on the CT scanning table while a radiologist moves a biopsy needle through the skin and toward the location of the mass. CT scans are repeated until the needle is within the mass. A biopsy sample is then removed to be looked at under a microscope.

Magnetic resonance imaging (MRI) scans : This test is not used as often as CT scans for lymphoma, but if doctor is concerned about spread to the spinal cord or brain, MRI is very useful for looking at these areas. Like CT scans, MRI scans provide detailed images of soft tissues in the body. But MRI scans use radio waves and strong magnets instead of x-rays. The energy from the radio waves is absorbed by the body and then released in a pattern formed by the type of body tissue and by certain diseases. A computer translates the pattern into a very detailed image of parts of the body. A contrast material called gadolinium may be injected into a vein before the scan to better see details. This material is different from what is used for CT scans, so being allergic to one doesn't mean you are allergic to the other. This material should be used with caution (if at all) in people on dialysis (for kidney failure).

Ultrasound : Ultrasound uses sound waves and their echoes to produce a picture of internal organs or masses. In the most common type of ultrasound, a small, microphone-like instrument called a transducer is placed on the skin (which is first lubricated with a gel). It emits sound waves and picks up the echoes as they bounce off the organs. The echoes are converted by a computer into a black and white image that is displayed on a computer screen. Ultrasound can be used to look at lymph nodes near the surface of the body or to look inside your abdomen for enlarged lymph nodes or organs such as the liver and spleen. It can also detect kidneys that have become swollen because the outflow of urine has been blocked by enlarged lymph nodes. (It can't be used to look at organs or lymph nodes in the chest because the ribs block the sound waves). This is an easy test to have done, and it uses no radiation. For most ultrasounds, simply lie on a table and a technician moves the transducer over the part of body being looked at.

Positron emission tomography (PET) scan : For a PET scan, a form of radioactive sugar (known as fluorodeoxyglucose or FDG) is injected into the blood. Because cancer cells in the body grow rapidly, they absorb large amounts of the radioactive sugar. After about an hour, will be moved onto a table in the PET scanner, lie on the table for about 30 minutes while a special camera creates a picture of areas of radioactivity in the body. The picture is not finely detailed like a CT or MRI scan, but it can provide helpful information about whole body. PET scans can help tell if an enlarged lymph node contains lymphoma. It can also help spot small areas that might be lymphoma, even if the area looks normal on a CT scan. PET scans can be used to tell if a lymphoma is responding to treatment. Some doctors will repeat

the PET scan after 1 or 2 courses of chemotherapy. If the chemotherapy is working, the lymph nodes will no longer take up the radioactive sugar. PET scans can also be used after treatment in helping decide whether an enlarged lymph node still contains lymphoma or is merely scar tissue. Often, for patients with lymphoma, a machine that combines the PET scan with a CT scan (PET/CT scan) is used. This allows the doctor to compare areas of higher radioactivity on the PET scan with the more detailed appearance of that area on the CT.

The stage is based on where lymphoma cells are found (in the lymph nodes or in other organs or tissues). The stage also depends on how many areas are affected. The stages of non-Hodgkin lymphoma are as follows:

Stage I : The lymphoma cells are in one lymph node group (such as in the neck or underarm). Or, if the abnormal cells are not in the lymph nodes, they are in only one part of a tissue or organ (such as the lung, but not the liver or bone marrow).

Stage II : The lymphoma cells are in at least two lymph node groups on the same side of (either above or below) the diaphragm or, the lymphoma cells are in one part of an organ and the lymph nodes near that organ (on the same side of the diaphragm). There may be lymphoma cells in other lymph node groups on the same side of the diaphragm.

Stage III : The lymphoma is in lymph nodes above and below the diaphragm. It also may be found in one part of a tissue or an organ near these lymph node groups.

Stage IV : Lymphoma cells are found in several parts of one or more organs or tissues (in addition to the lymph nodes). Or, it is in the liver, blood, or bone marrow.

Recurrent : The disease returns after treatment. In addition to these stage numbers, doctor may also describe the stage as A or B:

A: Patients have not had weight loss, drenching night sweats, or fevers.

B: Patients have had weight loss, drenching night sweats or fevers.

Gallium scan : For this test, a solution containing slightly radioactive gallium is injected into a vein. It is attracted to lymph tissue in the body. A few days later a special camera is used detect the radioactivity, showing the location of the gallium. The gallium scan will not detect most slow-growing lymphomas but will find many fast-growing (aggressive) lymphomas. This test is not used as much now as in the past, as many doctors may do a PET scan instead. It can still sometimes be useful in finding areas of lymphoma that the PET scan may miss. It can also help distinguish an infection from a lymphoma when the diagnosis is not clear.

Bone scan : For bone scans, a radioactive substance called technetium is used. After being injected into a vein, it travels to damage areas of the bone. Lymphoma often causes bone damage, and a bone scan will find it. But a bone scan may also pick up non-cancerous problems, such as arthritis and fractures. This test is not usually done unless a person is having bone pain or has lab test results that suggest the lymphoma may have reached the bones.

Tests of heart and lung function : These tests are not used to help diagnose non-Hodgkin lymphoma, but they may be done if you are going to get certain chemotherapy

drugs commonly used to treat lymphoma that may affect the heart or the lungs.

Non-Hodgkin lymphoma diagnosis and treatment statistics : Diagnosis and treatment statistics for non-Hodgkin lymphoma (NHL) are presented here by NHL subtype. There are also a discussion on factors which affect treatment decisions and data on clinical trials. Patients with NHL can present with a range of symptoms, though most present with lymphadenopathy–painless lumps caused by enlargement of lymph nodes. NHL is confirmed, and the extent of the disease is ascertained, using a range of tests. Though national guidance recommends referral to specialist services for investigation of lymphadenopathy, there is evidence that only around 1 in 10 UK patients are referred directly to a haematology department, with many others referred initially to general surgery; ear, nose and throat (ENT); or accident and emergency (A&E) departments; this is associated with delays in diagnosis.

Treatments:

A number of different treatments may be available to patients with NHL. The goals of NHL therapy may be cure, disease control or symptom relief, with management generally depending on the type and extent of disease. The choice of treatment depends mainly on the following:

- The type of non-Hodgkin lymphoma (for example, follicular lymphoma)
- Its stage (where the lymphoma is found)
- How quickly the cancer is growing (whether it is indolent or aggressive lymphoma)
- Age
- Whether you have other health problems

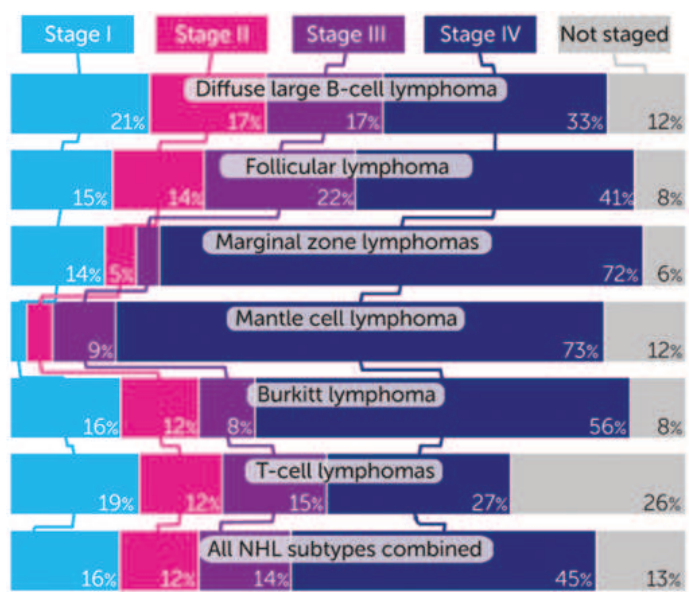


Figure : First-Line Treatments, Persons, HMRN, 2004-2011

Indolent NHL subtypes : For more indolent conditions, such as marginal zone lymphomas (MZL) and follicular lymphoma (FL), treatment may not be required if the patient is asymptomatic. Instead, patients are monitored for indicators of disease progression (an approach known as observation only, ‘watch and wait’, or ‘active monitoring’), and some patients will in fact never require treatment. Data from HMRN show that observation only is the first-line treatment decision for 45% of patients with MZL and 34% of those with FL. However, active treatment is also given in a substantial proportion of cases, for example within HMRN 32% of MZL patients and 51% of FL patients receive chemotherapy as their first treatment (Figure 5.1). Stage of disease may also influence treatment decisions, particularly in patients with Stage I disease. There is some evidence that Stage IA FL, for example, may be cured with radiotherapy; hence 11% of HMRN patients with FL receive first-line radiotherapy treatment. Radiotherapy may also be given in Stage I systemic MZL. Patients with *Helicobacter pylori*-associated gastric MZL, Stage IE, are usually treated solely with antibiotic eradication therapy. The majorities of patients, and particularly those with more aggressive disease, receive first-line chemotherapy treatment. Within HMRN, chemotherapy was the first treatment given to 79% of patients with diffuse large B-cell lymphoma (DLBCL) and 66% of those with mantle cell lymphoma (MCL). B-cell lymphoma chemotherapy is frequently combined with immunotherapy, such as the monoclonal antibody rituximab (Mabthera) for DLBCL and FL (Table 5.1).⁵ The International Prognostic Index (IPI)^{6,7} also influences treatment choice, with ‘high risk’ patients (high/high intermediate IPI) more likely to be entered into more aggressive chemotherapy trials. A small proportion (2%) of DLBCL patients receives radiotherapy: in patients with limited stage disease, involved field radiotherapy usually follows a short course of chemoimmunotherapy; whilst some patients with primary cerebral lymphoma receive whole brain radiotherapy. Asymptomatic MCL patients, especially the elderly and those with predominantly marrow-based disease, may be actively monitored; and this approach is used to manage 20% of patients within HMRN. In younger and/or fitter patients, more intensive regimes maybe used, with patients who respond being considered for autologous stem cell transplantation (using the patient’s own stem cells).

T-cell lymphomas : T-cell lymphomas are an extremely complex and heterogeneous group of diseases. The commonest subtype, peripheral T-cell lymphomas, is usually treated with chemotherapy, hence 59% of T-cell lymphoma patients in HMRN receive chemotherapy. Some will receive an autologous stem cell transplant, where possible. T-cell cutaneous (skin) lymphomas (mycosis fungoides and Sezary syndrome) are often treated with regimens that initially involve active monitoring, so this treatment accounts for 15% of T-cell lymphoma patients in HMRN. Patients with refractory (therapy-resistant) disease may subsequently receive photochemotherapy (PUVA, 7%), interferon, radiotherapy (3%) and alemtuzumab.

Factors affecting treatment decisions : Across all NHL subtypes, the patient’s ability to tolerate particular treatments will be incorporated into the decision-making process. Factors such as biological age and other illnesses impact on performance status and inevitably some

patients will be considered unable to withstand chemotherapy. First-line treatment for these patients may involve supportive care (such as blood transfusions) as well as a palliative approach; and within HMRN, 12% of patients are managed this way. This multi-disciplinary approach may involve additional support from clinical nurse specialists, specialist palliative care practitioners and psycho-oncologists. Treatment given for relapse will depend on several factors, including what first-line treatment the patient received.

- 1975 : Therapeutic antibodies are developed by Cesar Milstein and Georges Kohler.
- 1995 : Development and commercialisation of rituximab (US trade name Rituxan) begins in US.
- 1997 : US Food and Drug Administration (FDA) approves Rituxan for the treatment of relapsed or refractory low-grade NHL in the US.
- 1998 : European Medicines Agency (EMeA) authorises rituximab (European trade name MabThera) for the treatment of NHL in Europe.
- 2003 : National Institute for Health and Clinical Excellence (NICE) approves rituximab (only with CHOP) as first-line treatment for DLBCL stage II-IV in England and Wales (TA65).
- 2008 : NICE approves rituximab for remission induction/maintenance/last-line treatment for relapsed/refractory FL stage III/IV in England and Wales.
- 2009 : NICE approves rituximab (only with fludarabine and cyclophosphamide) as first-line treatment for CLL in England and Wales (TA174).
- 2010 : NICE approves rituximab (only with fludarabine and cyclophosphamide) for relapsed/refractory CLL in England and Wales (TA193).
- 2011 : NICE approves rituximab as maintenance therapy for FL which responded to first-line rituximab, in England and Wales (TA226).
- 2012 : NICE approves rituximab (only with CVP/CHOP/MCP/CHVPi/chlorambucil) as first-line treatment for symptomatic FL stage III/IV in England and Wales (TA243).
- 2012 : British Committee for Standards in Haematology (BCSH) recommends rituximab with chemotherapy for patients with MCL.

Clinical trials : The UK's National Cancer Research Network's Lymphoma Group Portfolio Maps for NHL currently list 30 trials which are open and recruiting: eleven for DLBCL; five for FL; three for MCL; and the remainder for other NHL subtypes or all NHL subtypes combined.¹² They also list 14 funded trials in the process of being set up: four each for DLBCL and FL; one for MCL; the remainder for other NHL subtypes or all NHL subtypes combined.¹² Most of these trials focus on first- or second-line treatments, and include: comparing different rituximab-chemotherapy combinations for older FL patients (PACIFICO); assessing the safety and efficacy of lower-intensity conditioning prior to allogeneic stem cell transplantation for MCL (Mini Allo); and evaluating the benefits of the addition of bortezomib to R-CHOP therapy for DLBCL (REMoDL-B). Recruitment of patients

to NHL trials is low in comparison with trials for solid tumours and other haematological cancers. In 2011-2012, an estimated 6% of cancer patients across all tumour sites participated in a randomised controlled trial (RCT) within the NIHR Clinical Research Network Portfolio. In contrast, in 2011-12 around 2% of eligible NHL patients were entered into an NIHR RCT.

Supportive Care : Non-Hodgkin lymphoma and its treatment can lead to other health problems. Patients may receive supportive care to prevent or control these problems and to improve comfort and quality of life during treatment. Patients may receive antibiotics and other drugs to help protect from infections. Patients' health care team may advise to stay away from crowds and from people with colds and other contagious diseases. If an infection develops, it can be serious, and will need treatment right away. Non-Hodgkin lymphoma and its treatment also can lead to anemia, which may make feel very tired. Drugs or blood transfusions can help with this problem.

Nutrition and Physical Activity : It's important for Patients' to take care of yourself by eating well and staying as active as you can. Patients' need the right amount of calories to maintain a good weight as well as needs enough protein to keep up strength. Eating well may help Patients' feel better and have more energy. Sometimes, especially during or soon after treatment, Patients' may not feel like eating, may be uncomfortable or tired. In addition, the side effects of treatment (such as poor appetite, nausea, vomiting, or mouth sores) can make it hard to eat well. Doctor, a registered dietitian, or another health care provider can suggest ways to deal with these problems. Many people find they feel better when they stay active. Walking, yoga, swimming, and other activities can keep strong and increase energy. Exercise may reduce nausea and pain and make treatment easier to handle. It also can help relieve stress.

Follow-up Care : Patients' need regular checkups after treatment for non-Hodgkin lymphoma. Doctor will watch patients' recovery closely and check for recurrence of the lymphoma. Checkups help make sure that any changes in health are noted and treated as needed. Checkups may include a physical exam, lab tests, chest x-rays, and other procedures. Between scheduled visits, patients' should contact the doctor right away if patients' have any health problems.

Sources of Support : Learning patients' have non-Hodgkin lymphoma can change life and the lives of those close to patients'. These changes can be hard to handle. It's normal for patients', patients' family, and patients' friends to have many different and sometimes confusing feelings. Concerns about treatments and managing side effects, hospital stays, and medical bills are common. Patients' also may worry about caring for patients' family, keeping job or continuing daily activities. Here's where you can go for support:

- Doctors, nurses, and other members of health care team can answer many of patients' questions about treatment, working, or other activities.
- Social workers, counselors, or members of the clergy can be helpful if patients' want to talk about patients' feelings or concerns. Often, social workers can suggest resources for

financial aid, transportation, home care, or emotional support.

- Support groups can also help. In these groups, patients or their family members meet with other patients or their families to share what they have learned about coping with the disease and the effects of treatment. Groups may offer support in person, over the telephone, or on the Internet.

Prognosis : The 5-year relative survival rate of patients with NHL is approximately 63%. The survival rate has steadily improved over the last 2 decades, thanks to improvements in medical and nursing care, the advent of novel therapeutic strategies (ie, monoclonal antibodies), validation of biomarkers of response, and the implementation of tailored treatment. The prognosis for patients with NHL depends on the following factors:

- Tumor histology (based on Working Formulation classification)
- Tumor stage
- Patient age
- Tumor bulk
- Performance status
- Serum lactate dehydrogenase (LDH) level
- Beta2-microglobulin level
- Presence or absence of extranodal disease

In general, these clinical characteristics are thought to reflect the following host or tumor characteristics:

- Tumor growth and invasive potential (eg, LDH, stage, tumor size, beta2-microglobulin level, number of nodal and extranodal sites, bone marrow involvement)
- Patient's response to tumor (eg, performance status, B symptoms)
- Patient's tolerance of intensive therapy (eg, performance status, patient age, bone marrow involvement).

The International Prognostic Index (IPI), which was originally designed as a prognostic factor model for aggressive NHL, also appears to be useful for predicting the outcome of patients with low-grade lymphoma and mantle cell lymphoma. This index is also used to identify patients at high risk of relapse, based on specific sites of involvement, including bone marrow, CNS, liver, testis, lung, and spleen. These patients may be considered for clinical trials that aim at improving the current treatment standard. An age-adjusted model for patients younger than 60 years has been proposed. In younger patients, stage III or IV disease, high LDH levels, and non-ambulatory performance status are independently associated with decreased survival rates. Pediatric and adolescent patients have better outcome than adults with CNS lymphoma. An ECOG performance status score of 0-1 is associated with improved survival. Higher dose methotrexate is associated with slightly better response. Clinical features included in the IPI that are independently predictive of survival include the following:

- Age - Younger than 60 years versus older than 60 years
- LDH level - Within the reference range versus elevated
- Performance status - Eastern Cooperative Oncology Group (ECOG) grade 0-1 versus 2-4
- Ann Arbor stage - Stage I-II versus III-IV
- Number of extranodal sites - Zero to 1 versus more than 1

With this model, relapse-free and overall survival rates at 5 years are as follows:

- risk factors - 75%
- 2-3 risk factors - 50%
- 4-5 risk factors - 25%

Patients with follicular lymphoma : The second most common subtype of NHL. The Follicular Lymphoma International Prognostic Index (FLIPI) score appears to be more discriminating than the IPI. The FLIPI score is calculated on the basis of 5 adverse prognostic factors, as follows:

- Age (>60 y)
- Ann Arbor stage (III-IV)
- Hemoglobin level (< 12 g/dL)
- Number of nodal areas (>4)
- Serum LDH level (above normal)

Some study proved that maternal smoking during pregnancy may have a modest increase in the risk for childhood NHL but not HL. Biomarkers in tumor cells such as the expression of bcl- 2 or bcl- 6 proteins and cDNA microarray provide useful prognostic information. Patients with congenital or acquired immunodeficiency have an increased risk of lymphoma and respond poorly to therapy. Time to achieve complete remission (CR) and response duration has prognostic significance. Patients who do not achieve CR by the third cycle of chemotherapy have a worse prognosis than those who achieve rapid CR. Patients with aggressive T- or NK-cell lymphomas generally have worse prognoses than those with B-cell lymphomas, except the Ki-1 anaplastic large T- or null-cell lymphomas. Cytogenetic abnormalities and oncogene expression affect prognosis. Patients with lymphomas with 1, 7, and 17 chromosomal abnormalities have worse prognoses than those with lymphomas without these changes. Low-grade lymphomas have indolent clinical behavior and are associated with a comparatively prolonged survival (median survival is 6-10 y), but they have little potential for cure when the disease manifests in more advanced stages. They also have the tendency to transform to high-grade lymphomas. Approximately 70% of all patients with intermediate and high-grade NHL relapse or never respond to initial therapy. Most recurrences are within the first 2 years after therapy completion. Patients with relapsed or resistant NHL have a very poor prognosis (< 5-10% are alive at 2 years with conventional

salvage chemotherapy regimens). Low levels of vitamin D were associated with a decrease in clinical end points (event-free survival and overall survival) in subsets of patients with aggressive B-cell lymphoma (ie, diffuse large B-cell lymphoma or T-cell lymphoma).

New research and treatment:

Research into the causes, prevention, and treatment of non-Hodgkin lymphoma is being done in many medical centers throughout the world. Scientists are making a lot of progress in understanding how changes in DNA can cause normal lymphocytes to develop into lymphoma cells. This is providing insight into why these cells may grow too rapidly, live too long, and not develop into mature cells that take part in normal immune reactions. Once this is understood, drugs may be developed that block this process. Progress in understanding DNA changes in lymphoma has already provided improved and highly sensitive tests for detecting this disease. Such tests can identify lymphoma cells based on changes such as chromosome translocations or rearrangements or specific gene mutations. Some of these tests are already in use, and others are being developed. They may be used to:

- Detect lymphoma cells in a biopsy sample
- Determine what type of lymphoma a person has
- Help determine if a lymphoma is likely to grow and spread, even within a certain subtype of lymphoma
- Help figure out if a certain treatment is likely to be helpful
- Help determine if a lymphoma has been destroyed by treatment and if a relapse is likely

Much of the research being done on non-Hodgkin lymphoma is focused on looking at new and better ways to treat this disease. Many new chemotherapy drugs are being studied in clinical trials. In recent years, these studies have led to the approval of drugs such as bendamustine (Treanda) and pralatrexate (Folotyn) for use against certain types of lymphoma. Other studies are looking at new ways to combine drugs using different doses or different sequences of drugs. Researchers continue to improve bone marrow and peripheral blood stem cell transplant methods, including new ways to collect these cells before the transplant. Autologous transplants (which use stem cells from the patient rather than from another person) have the risk of reintroducing lymphoma cells back into the patient after treatment. Researchers are testing new and improved ways to remove the last traces of lymphoma cells from the stem cells before they are returned to the patient. Some of the new monoclonal antibodies developed for treating lymphoma may help remove these remaining cells. A lot of research is focusing on eliminating graft-versus-host disease in allogeneic (donor) transplants. This work revolves around altering the transplanted T-cells so that they won't react with the recipient's normal cells but still kill the lymphoma cells. Researchers are also studying the effectiveness of non-myeloablative (reduced-intensity) stem cell transplants in people with lymphoma. This approach may allow more people to benefit from stem cell transplants. As researchers have learned more about cancer cells, they have developed newer

drugs that target specific parts of these cells. These are different from standard chemotherapy drugs, which work by attacking rapidly growing cells. The newer drugs often have different side effects, and they may work in some cases where chemotherapy doesn't. Targeted drugs such as bortezomib (Velcade), romidepsin (Istodax), and temsirolimus (Torisel) have shown some promise in treating certain lymphomas. These and similar drugs are now being studied in clinical trials.

Antibiotics : Gastric MALT lymphoma, which is linked to infection by the bacteria *Helicobacter pylori*, can often be treated with antibiotics against that bacterium. MALT lymphoma of the tissues around the eye (called ocular adnexal marginal zone lymphoma) has been linked to infection with the bacterium, *Chlamydia psittaci*. A recent study has shown that treating the infection with an antibiotic (doxycycline) can make this lymphoma get better and even go away. More studies may be needed before antibiotics become part of the standard treatment for this type of lymphoma.

Monoclonal antibodies : Lymphoma cells contain certain chemicals on their surface. Monoclonal antibodies that recognize these substances can be targeted to destroy the lymphoma cells while causing little damage to normal body tissues. This treatment strategy has already proven effective, such drugs, including rituximab, are already available in the market.

Immunotherapy : Rituximab is most often given for a limited amount of time during treatment. Because it has few side effects, it's been studied to see if using it long-term will help prevent lymphomas from coming back and help patients live longer. It does seem to help some patients with follicular lymphoma live longer, but using it long term for other lymphomas is still being studied. The success of rituximab and similar drugs such as ibritumomab and tositumomab, new monoclonal antibodies are being developed. Examples include epratuzumab, which targets the CD22 antigen on certain lymphoma cells, and obinutuzumab, which targets the CD20 antigen. Some newer antibodies are attached to substances that can poison cancer cells, and are known as immunotoxins. They act as homing devices to deliver the toxins directly to the cancer cells. One example of this is brentuximab vedotin (Adcetris), which is made up of an antibody to CD30 that is attached to a cell poison. It has been shown to help treat patients with anaplastic large cell lymphoma (ALCL) that is not responding to treatment with chemo. Another immunotoxin, known as CAT-3888 (BL22), targets the CD22 antigen on certain lymphoma cells, bringing along a toxin known as PE38. This drug showed a great deal of promise in treating hairy cell leukemia (HCL) in early clinical trials. A newer version of this drug, known as CAT-8015 (moxetumomab pasudotox), is now being studied for use against lymphomas.

Lymphoma vaccines : Some time people's immune systems may help fight their cancer. In rare instances, these people's immune systems have rejected their cancers, and they have been cured. Scientists are now trying to develop ways to encourage this immune reaction by using vaccines. Unlike vaccines against infections like measles or mumps, these vaccines are

designed to help treat, not prevent, lymphomas. The goal is to create an immune reaction against lymphoma cells in patients who have very early disease or in patients whose disease is in remission. One possible advantage of these types of treatments is that they seem to have very limited side effects. So far, there have been a few successes with this approach, and it's a major area of research in lymphoma treatment. At this time lymphoma vaccines are only available in clinical trials. BiovaxID™ is a vaccine based on the unique genetic makeup of a patient's B-cell non-Hodgkin lymphoma. The vaccine uses a unique protein (part of an antibody called an idiotypic) taken from each patient's own lymphoma cells, which are obtained during a biopsy. This protein is combined with substances that boost the body's immune response when the combination is injected into the patient. A late-stage clinical trial found that in people with follicular lymphomas that went away after chemotherapy, the vaccine lengthened the time before the lymphoma came back by more than a year. The vaccine has also shown promising early results against mantle cell lymphoma. It is not yet available outside of clinical trials.

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RADIATION THERAPY

Radiation therapy, radiotherapy, or radiation oncology, often abbreviated RT, RTx, or XRT treats cancer by using high energy to kill or control tumor or malignant cells without hurting healthy cells. Radiation therapy may be curative in a number of types of cancer if they are localized to one area of the body. Radiation can be given alone or used with other treatments, such as surgery or chemotherapy. It may also be used as part of adjuvant therapy, to prevent tumor recurrence after surgery to remove a primary malignant tumor (early stages of breast cancer). Radiation therapy is synergistic with chemotherapy, and has been used before, during, and after chemotherapy in susceptible cancers. In fact, certain drugs are known to be radio sensitizer's means they can actually make the cancer cells more sensitive to radiation, which helps the radiation to better, kill cancer cells. Like surgery, radiation therapy is a local treatment that affects cancer cells only in the treated area. Radiation can come from a machine (external radiation) or from a small container of radioactive material implanted directly into or near a tumor (internal radiation). External radiation therapy is usually given on an outpatient basis in a hospital or clinic. Patients are not radioactive during or after external radiation therapy. In case of internal radiation therapy, the patient stays in the hospital for a few days. The implant may be temporary or permanent. After an implant is removed, there is no radioactivity in the body. The amount of radiation in a permanent implant goes down to a safe level before the patient leaves the hospital. Side effects of radiation therapy depend on the treatment dose and the part of the body treated. The most common side effects of radiation are fatigue, skin reactions (such as a rash or redness) in the treated area, and loss of appetite. Radiation therapy can cause inflammation of tissues and organs in and around the body site that is radiated. Radiation therapy can also cause a decrease in the number of white blood cells. Although the side effects of radiation therapy can be unpleasant, they can usually be treated or controlled. Furthermore, in most cases, they are not permanent. Radiation oncology is the medical specialty concerned with prescribing radiation, and is distinct from radiology, the use of radiation in medical imaging and diagnosis. Radiation may be prescribed by a radiation oncologist with intent to cure ("curative") or for adjuvant therapy. It may also be used as palliative treatment (where cure is not possible and the aim is for local disease control or symptomatic relief) or as therapeutic treatment (where the therapy has survival benefit and it can be curative). It is also common to combine radiation therapy with surgery, chemotherapy, hormone therapy, immunotherapy or some mixture of the four. The precise treatment intent (curative, adjuvant, neoadjuvant, therapeutic, or palliative) will depend on the tumor type, location, and stage, as well as the general health of the patient. Total body irradiation (TBI) is a radiation therapy technique used to prepare the body to receive a bone marrow transplant. Brachytherapy, in which a radiation source is placed inside or next to the area requiring treatment, is another form of radiation therapy that minimizes exposure to healthy tissue during procedures to treat cancers of the breast, prostate and other

organs. Radiation therapy has several applications in non-malignant conditions, such as the treatment of trigeminal neuralgia, acoustic neuromas, severe thyroid eye disease, pterygium, pigmented villonodular synovitis, and prevention of keloid scar growth, vascular restenosis, and heterotopic ossification. The use of radiation therapy in non-malignant conditions is limited partly by worries about the risk of radiation-induced cancers.

Radiotherapy is usually required for one of the following reasons:

- stand-alone treatment to cure cancer
- to shrink a cancer before surgery
- during surgery to kill cancer cells that may remain in surrounding tissue after the surgery (called intraoperative radiation)
- after surgery to kill cancer cells remaining in the body
- to shrink an inoperable tumor in order to and reduce pain and improve quality of life
- to complement chemotherapy
- to control symptoms and improve quality of life if a cancer is too advanced to cure.

The history of radiotherapy :

Radiotherapy uses precisely targeted high-energy particles or waves such as x-rays, gamma rays, electron beams, or protons, to shrink tumors and destroy or damage cancer cells. It does this by damaging a cell's internal components (molecules), causing the cells to commit suicide by apoptosis. When a high-energy ray hits a molecule, it can cause it to break up. This can form free radicals, which can cause further damage to the inside of the cell. Some rare metals, for example radium, emit high-energy gamma-rays naturally. In other cases, radiation can be produced in a special machine, where a metal element is heated to produce electrons. These accelerate in an electric field towards a piece of tungsten metal, which then emits high-energy x-rays when they hit it.

Wilhelm Conrad Röntgen (27 March 1845 – 10 February 1923) was a German physicist, who, on 8 November 1895, produced and detected electromagnetic radiation in a wavelength range today that was known as X-rays or Röntgen rays, an achievement that earned him the first Nobel Prize in Physics in 1901. In honour of his accomplishments, in 2004 the International Union of Pure and Applied Chemistry (IUPAC) named element 111, roentgenium, a radioactive element with multiple unstable isotopes, after him. He gave a lecture called “Concerning a New Type Of Ray”, Roentgen called it the “X-ray”, with “x” being the algebraic symbol for an unknown quantity which caused a revolution in the scientific community. Just weeks later, Emil Grubbé, a student doctor in Chicago, became the first person to use of radiation to treat cancer. And three years later, two Swedish doctors used radiotherapy to cure several cases of head and neck cancer. In 1901, Röntgen was awarded the Nobel Prize for his discovery. The field of radiation therapy began to grow in the early 1900s largely due to the groundbreaking work of Nobel Prize-winning scientist Marie Curie (1867–1934), who discovered the radioactive elements polonium and radium in 1898. This

began a new era in medical treatment and research. Radium was used in various forms until the mid-1900s, when cobalt therapy and caesium units came into use. Finally, Medical linear accelerators have been used as radiation sources since the late 1940s. Early radiotherapy consisted of a single massive dose of radiation, typically lasting an hour. Side effects were severe. At the beginning of the 20th century, shortly after radiation began to be used for diagnosis and therapy, it was discovered that radiation could cause cancer as well as cure it. Many early radiologists used the skin of their arms to test the strength of radiation from their radiotherapy machines, looking for a dose that would produce a pink reaction (erythema) which looked like sunburn. They called this the “erythema dose,” and this was considered an estimate of the proper daily fraction of radiation. It’s no surprise that many of them developed leukemia from regularly exposing themselves to radiation. In 1914, an Austrian doctor controversially suggested that radiotherapy might work better if it was given in many smaller doses (‘fractionated radiotherapy’). This sparked a debate that lasted until 1922, when Claudius Regaud conclusively proved that fractionated therapy was just as effective as single-dose therapy, but caused fewer side effects. Despite these advances, radiotherapy was predominantly used to alleviate symptoms (‘palliative’ treatment) rather than actually cure cancer. Over the next thirty years, engineers built ever more powerful x-ray sources. The more powerful an x-ray penetrates further into the body and the less damage it does to the skin. X-ray energies are measured in Kilovolts (KV) or Megavolts (MV; $1\text{MV} = 1000\text{KV}$). In the 1920s, x-ray generators were only capable of making x-rays at about 200KV (for comparison, the natural gamma-rays emitted by radium are about 1.2MV). In the 1950s, engineers had developed an 8MV x-ray generator (or ‘linear accelerator’). Nowadays, an x-ray machine in a typical hospital will have energy of about 10MV. Since then, a number of technological developments have allowed radiologists to target the x-ray beam more accurately and avoid damaging normal tissue, further improving radiotherapy as a cancer treatment. Godfrey Hounsfield’s invention of computed tomography (CT) in 1971, three-dimensional planning became a possibility and created a shift from 2-D to 3-D radiation delivery. CT-based planning allows physicians to more accurately determine the dose distribution using axial tomographic images of the patient’s anatomy. Orthovoltage and cobalt units have largely been replaced by megavoltage linear accelerators, useful for their penetrating energies and lack of physical radiation source.

Advances in radiation physics and computer technology during the last quarter of the 20th century made it possible to aim radiation more precisely. Conformal radiation therapy (CRT) uses CT images and special computers to very precisely map the location of a cancer in 3 dimensions. The patient is fitted with a plastic mold or cast to keep the body part still and in the same position for each treatment. The radiation beams are matched to the shape of the tumor and delivered to the tumor from several directions. Intensity-modulated radiation therapy (IMRT) is like CRT, but along with aiming photon beams from several directions; the intensity (strength) of the beams can be adjusted. This gives even more control in decreasing the radiation reaching normal tissue while delivering a high dose to the cancer.

A related technique, conformal proton beam radiation therapy, uses a similar approach to focusing radiation on the cancer. This technique uses proton beams instead of using x-rays. Protons are parts of atoms that cause little damage to tissues they pass through but are very effective in killing cells at the end of their path. This means that proton beam radiation can deliver more radiation to the cancer while possibly reducing damage to nearby normal tissues. Stereotactic radio surgery and stereotactic radiation therapy are terms that describe several techniques used to deliver a large, precise radiation dose to a small tumor. The term surgery may be confusing because no cutting is actually done. The most common site treated with this radiation technique is the brain. A linear accelerator, or special machines such as the Gamma Knife or Cyber Knife, can be used to deliver this treatment. Intraoperative radiation therapy (IORT) is a form of treatment that delivers radiation at the time of surgery. The radiation can be given directly to the cancer or to the nearby tissues after the cancer has been removed. It's more commonly used in abdominal or pelvic cancers and in cancers that tend to recur (come back after treatment). IORT minimizes the amount of tissue that's exposed to radiation because normal tissues can be moved out of the way during surgery and shielded, allowing a higher dose of radiation to the cancer. Chemical modifiers or radiosensitizers are substances that make cancer more sensitive to radiation. The goal of research into these types of substances is to develop agents that will make the tumor more sensitive without affecting normal tissues. Researchers are also looking for substances that may help protect normal cells from radiation. For example, the use of CT scanning allows radiologists to determine the exact size and shape of the tumour. Whereas fractionated radiotherapy techniques such as CHART, developed by Cancer Research UK scientists in the 1990s, have been proved to increase survival and produce fewer side effects. Internal radiotherapy or brachytherapy involves implanting tiny beads or rods of radioactive metal around the tumour. It too has been in use since the early 1900s, and is still in use today. Initially, radium was the metal of choice for brachytherapy, but these days, caesium and iridium are used instead.

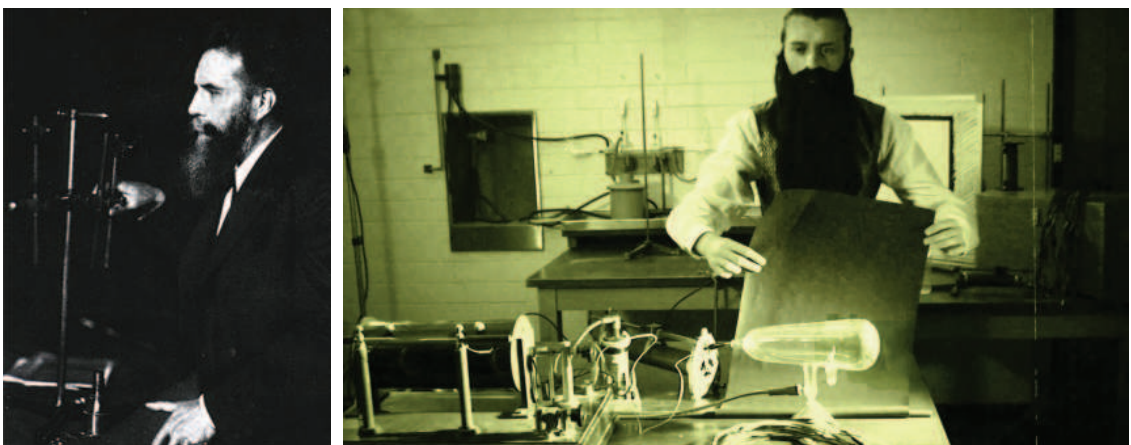


Fig: Wilhelm Conrad Röntgen.



Fig: World's First X-Ray (1895).

In 1895 Wilhelm Conrad Röntgen, a professor of physics Wurzburg University in Germany, to experiment with stopping power in the glass tube. In 1895 Wilhelm Röntgen conduct their own experiments, this time in the darkness and he is aware of the light generated in the wall, he knew not caused by fluorescence or visible light.

He found a particle beam “X” is not recognized, or better known as the X-ray. After several months playing with his invention he realizes where the paths of rays that form the image of the object, making it the first X-ray image in the world. In 1901 Wilhelm Röntgen was awarded the Nobel Prize for Physics for this discovery.

Types of radiation therapy:

Over 50% of cancer patients will undergo radiation therapy; for some, it will be the only cancer treatment they need. Radiation is often used in combination with other treatments. Used before or during other procedures, radiation shrinks the tumor to make surgery or chemotherapy more effective. Used afterward, it destroys any cancer cells that might remain. There are two basic types of radiation therapy namely:

- External beam radiation therapy
- Internal radiation or brachytherapy.

The differences relate to the position of the radiation source; external is outside the body, brachytherapy uses sealed radioactive sources placed precisely in the area under treatment. Brachytherapy can use temporary or permanent placement of radioactive sources. The temporary sources are usually placed by a technique called afterloading. In afterloading a hollow tube or applicator is placed surgically in the organ to be treated, and the sources are loaded into the applicator after the applicator is implanted. This minimizes radiation exposure to health care personnel. Particle therapy is a special case of external beam radiation therapy where the particles are protons or heavier ions. Intraoperative radiation therapy or IORT is a special type of radiation therapy that is delivered immediately after surgical removal of the cancer. This method has been employed in breast cancer, brain tumors and rectal cancers.

The radiation oncologist decides on the total dose of radiation that will be given to kill cancer cells and spare normal cells as much as possible. This dose is divided into a number of smaller doses called fractions. Fractionation schedules can vary. These are -

Standard (conventional) fractionation : The most common schedule for external beam radiation divides the total dose of radiation into several smaller doses or fractions. Treatments are usually given once a day, 5 days a week, and may last for several (about 3–8) weeks. **Hyperfractionation:** With hyperfractionated radiation, the daily dose of radiation is given over 2 or more sessions each day. The total period of time to complete the treatment schedule is not changed. In this radiation therapy using multiple smaller doses of radiation, a higher overall dose can be given.

Accelerated fractionation : With accelerated radiation therapy, the total dose of radiation is given over a shorter period of time by giving the same dose of radiation more than once a day. Accelerated fractionation does not change the total radiation dose.

Hypofractionation : With hypofractionated radiation therapy, fewer radiation treatments are given. This is done by giving either a short course of daily treatments or by giving fewer large doses (sometimes just a single treatment).

Boost : For some tumours, radiation may be given to a small area after the regular radiation treatment is finished. This radiation boost may be given externally or internally. It is used to reduce the risk of recurrence in a certain area. Radiation boosts can also be used to treat the tumour or area around the tumour with a higher dose than nearby normal tissues would tolerate.

External beam radiation therapy:

External beam radiation therapy is the most common kind of radiation therapy. It is usually done during outpatient visits to a hospital clinic and is usually covered by insurance. Once a radiation oncologist determines the proper dose of radiation for a particular cancer, the dose is divided into smaller doses called fractions. One fraction is usually given each day, five days a week for six to seven weeks. To minimize side effects, the treatments are typically given five days a week, Monday through Friday, for a number of weeks. This allows to get enough radiation into the body to kill the cancer while giving healthy cells time to recover. However, each radiation plan is individualized depending on the type and location of the cancer and what other treatments are also being used. The actual administration of the therapy usually takes about half an hour daily, although radiation is administered for only from one to five minutes at each session. It is important to attend every scheduled treatment to get the most benefit from radiation therapy. The type of machines used to administer external radiation therapy and the material that provides the radiation vary depending on the type and location of the cancer. Generally, the patient puts on a hospital gown and lies down or sits in a special chair. Parts of the body not receiving radiation are covered with special shields that block the rays. A technician then directs a beam of radiation to a pre-determined spot on the body where the cancer is located. The patient must stay still during the administration

of the radiation so that no other parts of the body are affected. As an extra precaution in some treatments, special molds are made to make sure the body is in the same position for each treatment. However, the treatment itself is painless, like having a bone x-rayed. During external beam radiation therapy, a beam (or multiple beams) of radiation is directed through the skin to the cancer and the immediate surrounding area in order to destroy the main tumor and any nearby cancer cells. The radiation beam is usually generated by a machine called a linear accelerator. The linear accelerator, or linac, is capable of producing high-energy X-rays or electrons for the treatment of cancer. Using treatment planning computers and software, treatment team controls the size and shape of the beam, as well as how it is directed at our body, to effectively treat our tumor while sparing the surrounding normal tissue.

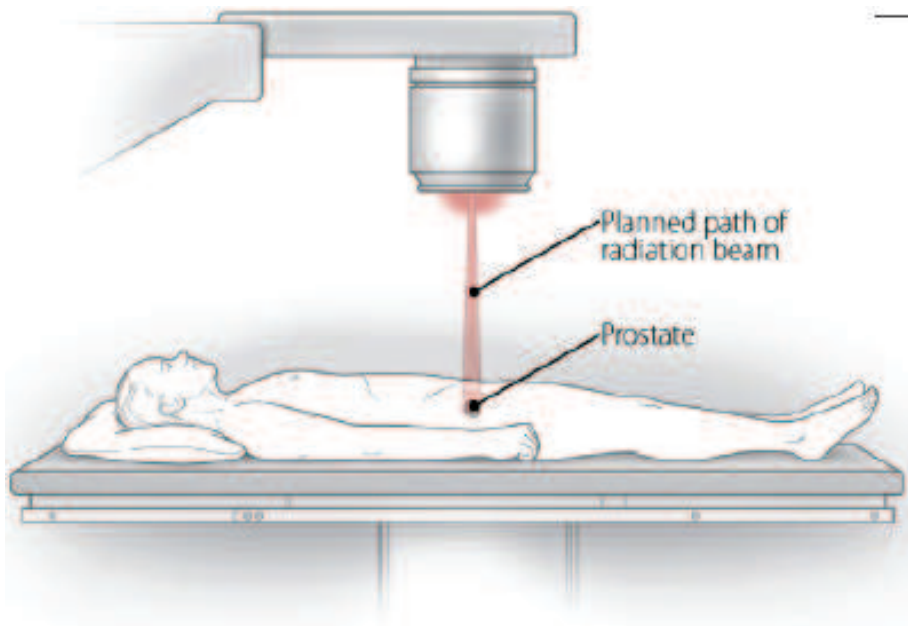


Fig: External beam radiation; During external beam radiation for prostate cancer, a patient will typically wear a gown or sweat pants that can easily be removed so that the area to be treated can be aligned with a ray of light that matches the path of the radiation. The radiation beam itself is not visible. The patient may also lie in a custom-made body “cast,” to immobilize the pelvis. Depending on the device used to deliver the radiation, each treatment takes about 15 to 20 minutes.

There are several special types of external beam therapy used for specific types of cancer, and radiation oncologist will recommend one of these treatments if he or she believes it will help the cancer patient.

Conventional external beam radiation therapy (2DXRT) : Conventional external beam radiation therapy (2DXRT) is delivered via two-dimensional beams using linear accelerator machines. 2DXRT mainly consists of a single beam of radiation delivered to the patient from several directions that is front or back, and both sides. Conventional refers to the way the treatment is planned or simulated on a specially calibrated diagnostic x-ray machine known as a simulator because it recreates the linear accelerator actions (or sometimes by

eye), and to the usually well-established arrangements of the radiation beams to achieve a desired plan. The aim of simulation is to accurately target or localize the volume which is to be treated. This technique is well established and is generally quick and reliable. The worry is that some high-dose treatments may be limited by the radiation toxicity capacity of healthy tissues which lay close to the target tumor volume. An example of this problem is seen in radiation of the prostate gland, where the sensitivity of the adjacent rectum limited the dose which could be safely prescribed using 2DXRT planning to such an extent that tumor control may not be easily achievable. Prior to the invention of the CT, physicians and physicists had limited knowledge about the true radiation dosage delivered to both cancerous and healthy tissue. For this reason, 3-dimensional conformal radiation therapy is becoming the standard treatment for a number of tumor sites.

Three-Dimensional Conformal Radiation Therapy (3D-CRT) : Three-dimensional conformal radiation therapy, or 3D-CRT, uses computers and special imaging techniques such as CT, MR or PET scans to show the size, shape and location of the tumor as well as surrounding organs. Radiation oncologist can then precisely tailor the radiation beams to the size and shape of tumor with multileaf collimators or custom fabricated field-shaping blocks. The radiation beams are very precisely directed, nearby normal tissue receives less radiation and is able to heal more quickly.

Intensity Modulated Radiation Therapy (IMRT) : Intensity modulated radiation therapy, or IMRT, is a specialized form of 3D-CRT that allows radiation to be more exactly shaped to fit the tumor. In this therapy the radiation beam can be broken up into many “beamlets,” and the intensity of each beamlet can be adjusted individually. Using IMRT, it may be possible to further limit the amount of radiation received by healthy tissue near the tumor. In some situations, this may also safely allow a higher dose of radiation to be delivered to the tumor, potentially increasing the chance of a cure.

Conformal proton beam radiation therapy : Conformal proton beam radiation therapy is much like conformal therapy, but it uses proton beams instead of x-rays. Protons are parts of atoms that cause little damage to tissues they pass through but are very good at killing cells at the end of their path. This means that proton beam radiation may be able to deliver more radiation to the tumor while reducing side effects on normal tissues. Protons can only be put out by a special machine called a cyclotron or synchrotron. This machine costs millions of dollars and requires expert staff. This is why proton beam therapy costs a lot and is only in a small number of radiation treatment centers.

Intraoperative radiation therapy : Intraoperative radiation therapy (IORT) is external radiation given directly to the tumor or tumors during surgery. It may be used if the tumors can't be removed completely or if there's a high risk the cancer will come back in the same area. The surgeon finds the cancer while the patient is under anesthesia drugs are used to make the patient sleep and not feel pain. Normal tissues are moved out of the way and protected with special shields, so IORT lets the doctor give one large dose of radiation to the cancer and

limit the effects on nearby tissues. IORT is usually given in a special operating room that has radiation-shielding walls.

Stereotactic radiosurgery:

Stereotactic radiosurgery is not really surgery, but a type of radiation treatment that gives a large dose of radiation to a small tumor area, usually in one session. It's mostly used for brain tumors and other tumors inside the head. In some cases, a head frame or shell may be used to help keep the patient's head still. Once the exact location of the tumor is known from CT or MRI scans, radiation is sent to the area from a machine. The radiation is very precisely aimed to affect nearby tissues as little as possible. There are 3 different ways stereotactic radiosurgery can be given:

- The most common type uses a movable linear accelerator that's controlled by a computer. The machine moves around to target the tumor from many different angles. Several machines do stereotactic radiosurgery in this way, with names such as X-Knife™, CyberKnife®, and Clinac®.
- The Gamma Knife® uses about 200 small beams aimed at the tumor from different angles for a short period of time to deliver a large dose of radiation. It's usually given in one treatment session. Again, it does not use a knife and there's no cutting.
- A third type uses heavy charged particle beams (like protons or helium ion beams) to deliver radiation to the tumor. The tumor from different angles, the particles allow most of the radiation's energy to be delivered to more precise depths, at the end of their paths. This limits damage to nearby healthy tissues or organs.

Most of the time, stereotactic radiosurgery uses one session to give the whole radiation dose, but it may be repeated if needed. Sometimes doctors give the radiation in many smaller treatments to deliver the same or slightly higher dose. This may be called fractionated radiosurgery or fractionated stereotactic radiotherapy.

Neutron Beam Therapy : Like proton therapy, neutron beam therapy is a specialized form of external beam radiation therapy. It is often used to treat certain tumors that are radioresistant, meaning they are very difficult to kill using conventional X-ray radiation therapy. Neutrons have a greater biologic impact on cells than other types of radiation. Used carefully, this added impact can be an advantage in certain situations. Neutron therapy is available at only a few centers in the country.

Image Guided Radiation Therapy (IGRT) : Radiation oncologists use image guided radiation therapy, or IGRT, to help better deliver the radiation to the cancer since tumors can move between treatments due to differences in organ filling or movements while breathing. IGRT involves conformal radiation treatment guided by imaging, such as CT, ultrasound or X-rays, taken in the treatment room just before the patient is given the radiation treatment on a daily basis.

All patients first undergo a CT scan as part of the planning process. The information

from the CT scan is then transmitted to a computer in the treatment room to allow doctors to compare the earlier image with the images taken just before treatment. During IGRT, doctors compare these images to see if the treatment needs to be adjusted. This allows doctors to better target the cancer while avoiding nearby healthy tissue. In some cases, doctors will implant a tiny marker in or near the tumor to pinpoint it for IGRT. This helps to account for organ/tumor motion even if the body is immobilized by a casting device.

Internal radiation therapy:

Internal radiation therapy is called brachytherapy, implant therapy, interstitial radiation, or intracavitary radiation. With internal radiation therapy, a bit of radioactive material is sealed in an implant (sometimes called a seed or capsule). The implant is then placed very close to the cancer. The advantage of internal radiation therapy is that it concentrates the radiation near the cancer and lessens the chance of damage to normal cells. Many different types of radioactive materials can be used in the implant, including cesium, iridium, iodine, phosphorus, and palladium. Internal radiation therapy is used for some cancers of the head, neck, thyroid, breast, female reproductive system, and prostate. Most people will have the radioactive capsule implanted by a surgeon while under either general or local anesthesia at a hospital or surgical clinic. Patients receiving internal radiation therapy do become temporarily radioactive. They must remain in the hospital during the time that the implant stays in place. The length of time is determined by the type of cancer and the dose of radioactivity to be delivered. During the time the implant is in place, the patient will have to stay in bed and remain reasonably still.

While the implant is in place, the patient's contact with other people will be limited. Healthcare workers will make their visits as brief as possible to avoid exposure to radiation, and visitors, especially children and pregnant women, will be limited. The implant usually can be removed in a simple procedure without an anesthetic. As soon as the implant is out of the body, the patient is no longer radioactive, and restrictions on being with other people are lifted. Generally people can return to a level of activity that feels comfortable to them as soon as the implant is removed. Occasionally the site of the implant is sore for some time afterwards. This discomfort may limit specific activities. In some cases, an implant is left permanently inside the body. People who have permanent implants need to stay in the hospital and away from other people for the first few days. Gradually the radioactivity of the implant decreases, and it is safe to be around other people. Brachytherapy is used to treat cancers throughout the body, including the prostate, cervix, head and neck, skin, breast, gallbladder, uterus, vagina, lung, rectum and eye.

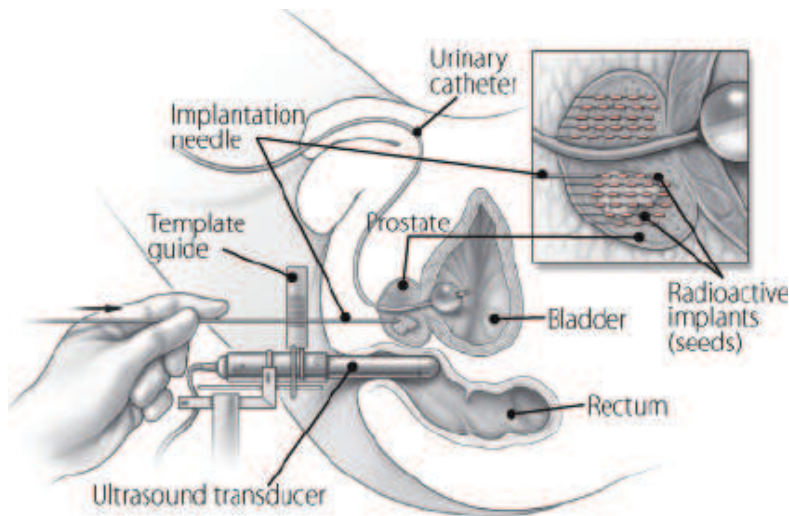


Figure 2: Brachytherapy; Most radiation oncologists use 3D treatment planning and a template guide to precisely implant radioactive seeds and to ensure that radiation is distributed evenly throughout the prostate. Ultrasound, delivered through an ultrasound probe, or transducer, allows them to view the prostate throughout the procedure.

The main types of brachytherapy are intracavitary radiation and interstitial radiation. Both of these methods use radioactive implants such as pellets, seeds, ribbons, wires, needles, capsules, balloons, or tubes.

- During intracavitary radiation, the radioactive source is placed in a cavity (space) in the body, such as the rectum or uterus.
- With interstitial radiation, the implants are placed in or near the cancer, but not in a body cavity.

Brachytherapy may be either temporary or permanent. In temporary brachytherapy, a highly radioactive material is placed inside a catheter or slender tube for a specific amount of time and then withdrawn. Temporary brachytherapy can be administered at a low-dose rate (LDR) or high-dose rate (HDR). Permanent brachytherapy, also called seed implantation, involves placing radioactive seeds or pellets (about the size of a grain of rice) in or near the tumor and leaving them there permanently. After several months, the radioactivity level of the implants eventually diminishes to nothing. The inactive seeds then remain in the body, with no lasting effect on the patient. Sometimes, these inactive metallic seeds can trigger metal detectors at airport security checkpoints.

Conventional radiation techniques such as external beam radiotherapy (EBRT) following surgical removal of the tumor have several drawbacks. The tumor bed where the highest dose should be applied is frequently missed due to the complex localization of the wound cavity even when modern radiotherapy planning is used. Additionally, the usual delay between the surgical removal of the tumor and EBRT may allow a repopulation of the tumor cells. These potentially harmful effects can be avoided by delivering the radiation more precisely to the

targeted tissues leading to immediate sterilization of residual tumor cells. Another aspect is that wound fluid has a stimulating effect on tumor cells. IORT was found to inhibit the stimulating effects of wound fluid.

Radioisotope therapy (RIT) : Systemic radioisotope therapy is a form of targeted therapy. Targeting can be due to the chemical properties of the isotope such as radioiodine which is specifically absorbed by the thyroid gland, a thousand fold better than other bodily organs. Targeting can also be achieved by attaching the radioisotope to another molecule or antibody to guide it to the target tissue. The radioisotopes are delivered through infusion (into the bloodstream) or ingestion. Examples are the infusion of metaiodobenzylguanidine (MIBG) to treat neuroblastoma, of oral iodine-131 to treat thyroid cancer or thyrotoxicosis, and of hormone-bound lutetium-177 and yttrium-90 to treat neuroendocrine tumors (peptide receptor radionuclide therapy). Another example is the injection of yttrium-90 radioactive glass or resin microspheres into the hepatic artery to radioembolize liver tumors or liver metastases. These microspheres are used for the treatment approach known as selective internal radiation therapy. The microspheres are approximately 30 μm in diameter (about one-third of a human hair) and are delivered directly into the artery supplying blood to the tumors. These treatments begin by guiding a catheter up through the femoral artery in the leg, navigating to the desired target site and administering treatment. The blood feeding the tumor will carry the microspheres directly to the tumor enabling a more selective approach than traditional systemic chemotherapy. A major use of systemic radioisotope therapy is in the treatment of bone metastasis from cancer. The radioisotopes travel selectively to areas of damaged bone, and spare normal undamaged bone. Isotopes commonly used in the treatment of bone metastasis are strontium-89 and samarium (^{153}Sm) lexidronam. In 2002, the United States Food and Drug Administration (FDA) approved ibritumomab tiuxetan (Zevalin), which is an anti-CD20 monoclonal antibody conjugated to yttrium-90. In 2003, the FDA approved the tositumomab/iodine (^{131}I) tositumomab regimen (Bexxar), which is a combination of an iodine-131 labelled and an unlabelled anti-CD20 monoclonal antibody. These medications were the first agents of what is known as radioimmunotherapy, and they were approved for the treatment of refractory non-Hodgkins lymphoma.

Radioimmunotherapy:

Radioimmunotherapy is a promising way to treat cancer that has spread (metastasized) to multiple locations throughout the body. Antibodies are immune system proteins that specifically recognize and bind to only one type of cell. They can be designed to bind only with a certain type of cancer cell. To carry out radioimmunotherapy, antibodies with the ability to bind specifically to a patient's cancer cells are attached to radioactive material and injected into the patient's bloodstream. When these man-made antibodies find a cancer cell, they bind to it. Then the radiation kills the cancer cell. This process is still experimental, but because it can be used to selectively attack only cancer cells, it holds promise for eliminating cancers that have spread beyond the primary tumor.

Radiation used to treat cancer:

Radiation used for cancer treatment is called ionizing radiation because it forms ions (electrically charged particles) in the cells of the tissues it passes through. It creates ions by removing electrons from atoms and molecules. This can kill cells or change genes so the cells stop growing. Other forms of radiation such as radio waves, microwaves, and light waves are called non-ionizing. They don't have as much energy and are not able to form ions. Ionizing radiation can be sorted into 2 major types:

- Photons (x-rays and gamma rays), which are most widely used in cancer treatment
- Particle radiation (such as electrons, protons, neutrons, carbon ions, alpha particles, and beta particles).

Some types of ionizing radiation have more energy than others. The higher the energy, the more deeply the radiation can penetrate (get into) the tissues. The way a certain type of radiation behaves is important in planning radiation treatments. The radiation oncologist selects the type and energy of radiation that is most suitable for each patient's cancer and location.

Photon radiation : The most common form of radiation used for cancer treatment is a high-energy photon beam. This comes from radioactive sources such as cobalt, cesium, or a machine called a linear accelerator (or linac, for short). Photon beams of energy affect the cells along their path as they go through the body to get to the cancer, pass through the cancer, and exit the body.

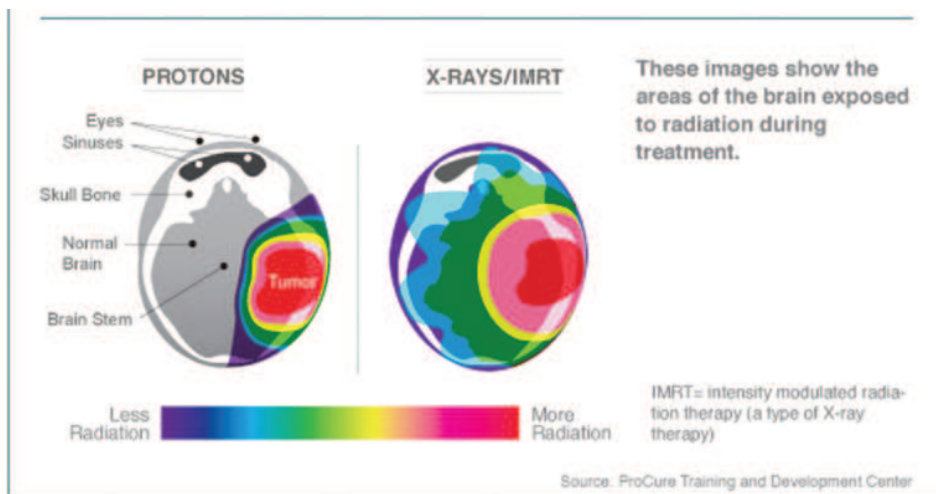


Fig: Proton therapy delivers less radiation to the brain stem, eyes and healthy tissue than conventional radiation, reducing the likelihood of side effects.

Particle radiation : Electron beams or particle beams are also produced by a linear accelerator. Electrons are negatively charged parts of atoms. They have a low energy level and don't penetrate deeply into the body, so this type of radiation is used most often to treat the skin, tumors, and lymph nodes that are close to the surface of the body. Proton beams are a

form of particle beam radiation. Protons are positively charged parts of atoms. They cause less damage to tissues they pass through but are very good at killing cells at the end of their path. Because of this, proton beams are thought to be able to deliver more radiation to the cancer while causing fewer side effects to normal tissues. Protons are used routinely for certain types of cancer, but still need more study in treating others.

Some of the techniques used in proton treatment can also expose the patient to neutrons. Neutron beams are used for some cancers of the head, neck, and prostate and for certain inoperable tumors. A neutron is a particle in many atoms that has no charge. Neutron beam radiation can sometimes help when other forms of radiation therapy don't work. Because neutrons can damage DNA more than photons, effects on normal tissue can be more severe. Beams must be aimed carefully and normal tissue protected. Neutron beams show great promise with salivary gland cancers that can't be cured with surgery.

Carbon ion radiation is also called heavy ion radiation because it uses a particle that is heavier than a proton or neutron. The particle is part of the carbon atom, which itself contains protons, neutrons, and electrons. Due to its so heavy nature, it can do more damage to the target cell than other types of radiation. As with protons, the beam of carbon ions can be adjusted to do the most damage to the cancer cells at the end of its path but the effects on nearby normal tissue can be more severe. This type of radiation is only available in a few centers in the world. It can be helpful in treating cancers that don't usually respond well to radiation (called radio resistant).

Alpha and beta particles are mainly produced by special radioactive substances that may be injected, swallowed, or put into the body of a person with cancer. They're most often used in imaging tests, but can be helpful in treating cancer.

Mechanism of radiation therapy:

Radiation therapy uses a special kind of high-energy beam to damage cancer cells. These high-energy beams, which are invisible to the human eye, damage a cell's DNA (the molecules inside cells that carry genetic information and pass it from one generation to the next). Radiation therapy can either damage DNA directly or create charged particles (free radicals) within the cells that can in turn damage the DNA. Cancer cells whose DNA is damaged beyond repair stop dividing or die. The damage is caused by a photon, electron, proton, neutron, or ion beam directly or indirectly ionizing the atoms which make up the DNA chain. Indirect ionization happens as a result of the ionization of water, forming free radicals, notably hydroxyl radicals, which then damage the DNA. In the most common forms of radiation therapy, most of the radiation effect is through free radicals because cells have mechanisms for repairing damage DNA. Breaking the DNA on both strands proves to be the most significant technique in modifying cell characteristics. Cancer cells generally are undifferentiated and stem cell-like, they reproduce more, and have a diminished ability to repair sub-lethal damage compared to most healthy differentiated cells. The DNA damage is inherited through cell division, accumulating damage to the cancer cells, causing them to die or reproduce more slowly. When the damaged cells die, they are broken down and eliminated

by the body's natural processes. The radiation damages cells that are in the path of its beam means radiation damages normal cells as well as cancer cells but radiation affects cancer cells more than normal cells. Cancer cells are less organized than healthy cells; it's harder for them to repair the damage done by radiation. So cancer cells are more easily destroyed by radiation, while healthy, normal cells are better able to repair themselves and survive the treatment. One of the major limitations of radiotherapy is that the cells of solid tumors become deficient in oxygen. Solid tumors can outgrow their blood supply, causing a low-oxygen state known as hypoxia. Oxygen is a potent radiosensitizer, increasing the effectiveness of a given dose of radiation by forming DNA-damaging free radicals. Tumor cells in a hypoxic environment may be as much as 2 to 3 times more resistant to radiation damage than those in a normal oxygen environment. Much research has been devoted to overcoming this problem including the use of high pressure oxygen tanks, blood substitutes that carry increased oxygen, hypoxic cell radiosensitizers such as misonidazole and metronidazole, and hypoxic cytotoxins, such as tirapazamine. There is also interest in the fact that high-LET (linear energy transfer) particles such as carbon or neon ions may have an antitumor effect which is less dependent of tumor oxygen because these particles act mostly via direct damage.

There are two different ways to deliver radiation to the tissues to be treated:

- A machine called a linear accelerator that delivers radiation from outside the body
- Pellets, or seeds, of material that give off radiation beams from inside the body. Tissues to be treated might include the breast area, lymph nodes, or another part of the body.

In some cases, oncologist may recommend hyperthermia be used in combination with radiation therapy. Hyperthermia (also called thermal therapy or thermotherapy) uses an energy source such as ultrasound or microwave to heat cancer cells to high temperatures, up to 113 degrees Fahrenheit. Early research has shown that hyperthermia may make some cancer cells more sensitive to radiation. Hyperthermia is still being studied in clinical trials and isn't available everywhere. Hyperthermia and radiation are usually given within an hour of each other.

Some people may worry that therapeutic radiation may be dangerous like an atomic bomb or nuclear power plant. Stories about radiation side effects, some of them exaggerated can circulate around hospital waiting rooms. It's important to know that there is NO connection between therapeutic radiation and the types of radiation in bombs and nuclear reactors. The radiation used in cancer treatment is highly focused, controllable, and generally safe.

Importance of radiation therapy:

Radiation is an important and often necessary form of anti-cancer therapy because it is able to reduce the risk of recurrence after surgery means removed all the cancer including breast cancer. Surgery cannot guarantee that every last cancer cell has been removed from the body. Individual cancer cells are too small to be felt or seen during surgery or detected by testing. Any cells that remain after surgery can grow and eventually form a new lump or show up as an abnormality on a test such as a mammogram. Research has shown that people

who are treated with radiation after lumpectomy are more likely to live longer, and remain cancer-free longer, than those who don't get radiation. In one large study, women who didn't get radiation after lumpectomy were shown to have a 60% greater risk of the cancer coming back in the same breast. Other research has shown that even women with very small cancers (1 centimeter or smaller) benefit from radiation after lumpectomy. Radiation therapy may also be given with palliative intent. Palliative treatments are not intended to cure. Instead, they relieve symptoms and reduce the suffering caused by cancer. Some examples of palliative radiation therapy are:

- Radiation given to the brain to shrink tumors formed from cancer cells that have spread to the brain from another part of the body (metastases).
- Radiation given to shrink a tumor that is pressing on the spine or growing within a bone, which can cause pain.
- Radiation given to shrink a tumor near the esophagus, which can interfere with a patient's ability to eat and drink.

When a patient get radiation therapy:

A patient may receive radiation therapy before, during, or after surgery. Some patients may receive radiation therapy alone, without surgery or other treatments whereas some patients may receive radiation therapy and chemotherapy at the same time. The timing of radiation therapy depends on the type of cancer being treated and the goal of treatment (cure or palliation). Radiation therapy given before surgery is called pre-operative or neoadjuvant radiation. Neoadjuvant radiation may be given to shrink a tumor so it can be removed by surgery and be less likely to return after surgery. Radiation therapy given during surgery is called intra-operative radiation therapy (IORT). IORT is sometimes used when normal structures are too close to a tumor to allow the use of external-beam radiation therapy. Radiation therapy given after surgery is called post-operative or adjuvant radiation therapy. Radiation therapy given after some types of complicated surgery (especially in the abdomen or pelvis) may produce too many side effects; therefore, it may be safer if given before surgery in these cases. The combination of chemotherapy and radiation therapy given at the same time is sometimes called chemoradiation or radiochemotherapy. The combination of chemotherapy and radiation therapy may kill more cancer cells (increasing the likelihood of a cure), but it can also cause more side effects. After cancer treatment, patients receive regular follow-up care from their oncologists to monitor their health and to check for possible cancer recurrence.

Treatment of radiation therapy for an individual patient:

A radiation oncologist develops a patient's treatment plan through a process called treatment planning, which begins with simulation. During simulation, detailed imaging scans show the location of a patient's tumor and the normal areas around it. These scans are usually computed tomography (CT) scans, but they can also include magnetic resonance imaging (MRI), positron emission tomography (PET), and ultrasound scans. CT scans are

often used in treatment planning for radiation therapy. During CT scanning, pictures of the inside of the body are created by a computer linked to an x-ray machine.

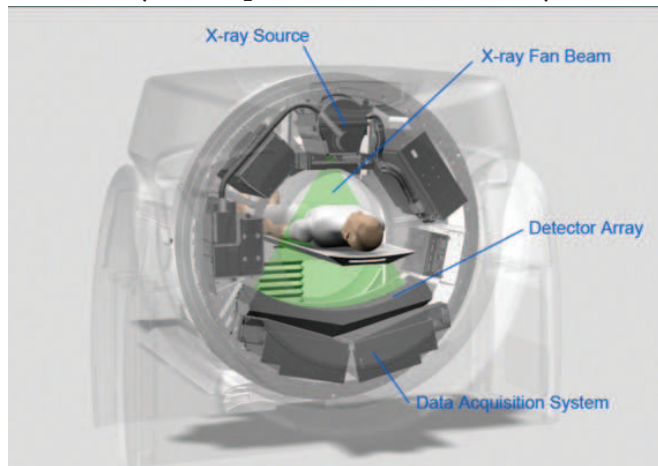


Fig: Computed Tomography Scanner. CT scans are often used in treatment planning for radiation therapy. During CT scanning, pictures of the inside of the body are created by a computer linked to an x-ray machine.

During simulation and daily treatments, it is necessary to ensure that the patient will be in exactly the same position every day relative to the machine delivering the treatment or doing the imaging. Body molds, head masks, or other devices may be constructed for an individual patient to make it easier for a patient to stay still. Temporary skin marks and even tattoos are used to help with precise patient positioning. Patients getting radiation to the head may need a mask. The mask helps keep the head from moving so that the patient is in the exact same position for each treatment.



Fig: In order to include the exact tumor volume, and also to protect surrounding tissue, specialized positioning devices, or masks for head and neck areas, are employed. Small marks are applied directly to the skin, or to the mask, in order to ensure the exact same positioning and settings for each daily treatment.

After simulation, the radiation oncologist then determines the exact area that will be treated, the total radiation dose that will be delivered to the tumor, how much dose will be allowed for the normal tissues around the tumor, and the safest angles (paths) for radiation

delivery. The staff working with the radiation oncologist use sophisticated computers to design the details of the exact radiation plan that will be used. After approving the plan, the radiation oncologist authorizes the start of treatment. On the first day of treatment, and usually at least weekly after that, many checks are made to ensure that the treatments are being delivered exactly the way they were planned. Radiation doses for cancer treatment are measured in a unit called a gray (Gy), which is a measure of the amount of radiation energy absorbed by 1 kilogram of human tissue. Different doses of radiation are needed to kill different types of cancer cells. Radiation can damage some types of normal tissue more easily than others. For example, the reproductive organs (testicles and ovaries) are more sensitive to radiation than bones. If an area of the body has previously been treated with radiation therapy, a patient may not be able to have radiation therapy to that area a second time, depending on how much radiation was given during the initial treatment. If one area of the body has already received the maximum safe lifetime dose of radiation, another area might still be treated with radiation therapy if the distance between the two areas is large enough. The area selected for treatment usually includes the whole tumor plus a small amount of normal tissue surrounding the tumor. The normal tissue is treated for two main reasons:

- To take into account body movement from breathing and normal movement of the organs within the body, this can change the location of a tumor between treatments.
- To reduce the likelihood of tumor recurrence from cancer cells those have spread to the normal tissue next to the tumor (called microscopic local spread).

Patient's condition and radiation therapy:

Radiation can come from a machine outside the body (external-beam radiation therapy) or from radioactive material placed in the body near cancer cells (internal radiation therapy, more commonly called brachytherapy). Systemic radiation therapy uses a radioactive substance, given by mouth or into a vein that travels in the blood to tissues throughout the body. The type of radiation therapy prescribed by a radiation oncologist depends on many factors, including:

- The type of cancer
- The size of the cancer
- The cancer's location in the body
- How close the cancer is to normal tissues that are sensitive to radiation
- How far into the body the radiation needs to travel
- The patient's general health and medical history
- Whether the patient will have other types of cancer treatment
- Other factors, such as the patient's age and other medical conditions.

Effect of radiation therapy on different types of cancer:

The response of a cancer to radiation is described by its radio sensitivity. Highly

radiosensitive cancer cells such as leukemias, most lymphomas and germ cell tumors are rapidly killed by modest doses of radiation. To achieve a radical cure, the majorities of epithelial cancers are only moderately radiosensitive, and require a significantly higher dose of radiation (60-70 Gy). Some types of cancer are notably radio resistant means much higher doses are required to produce a radical cure than may be safe in clinical practice. Renal cell cancer and melanoma are generally considered to be radio resistant. It is important to distinguish the radio sensitivity of a particular tumor, which to some extent is a laboratory measure, from the radiation “curability” of a cancer in actual clinical practice. For example, leukemias are not generally curable with radiation therapy, because they are disseminated through the body. Lymphoma may be radically curable if it is localized to one area of the body. Similarly, many of the common, moderately radio responsive tumors are routinely treated with curative doses of radiation therapy if they are at an early stage. For example: non-melanoma skin cancer, head and neck cancer, breast cancer, non-small cell lung cancer, cervical cancer, anal cancer, prostate cancer. Metastatic cancers are generally incurable with radiation therapy because it is not possible to treat the whole body. The response of a tumor to radiation therapy is also related to its size. For complex reasons, very large tumors respond less well to radiation than smaller tumors or microscopic disease. Various strategies are used to overcome this effect. The most common technique is surgical resection prior to radiation therapy. This is most commonly seen in the treatment of breast cancer with wide local excision or mastectomy followed by adjuvant radiation therapy. Another method is to shrink the tumor with neoadjuvant chemotherapy prior to radical radiation therapy. A third technique is to enhance the radio sensitivity of the cancer by giving certain drugs during a course of radiation therapy. Examples of radio sensitizing drugs include: Cisplatin, Nimorazole, and Cetuximab. The effect of radiotherapy on control of cancer has been shown to be limited to the first five years after surgery, particularly for breast cancer. The difference between breast cancer recurrences in patients who receive radiotherapy vs. those who don't is seen mostly in the first 2–3 years and no difference is seen after 5 years.

Common side effects of radiation therapy:

Radiotherapy is an effective treatment for many cancers, but it can cause side effects. People react quite differently to radiotherapy, and some people may have no side effects. Radiation therapy can cause both early (acute) and late (chronic) side effects. Acute side effects occur during treatment, and chronic side effects occur months or even years after treatment ends. The side effects that develop depend on the area of the body being treated, the dose given per day, the total dose given, the patient's general medical condition, and other treatments given at the same time. Acute radiation side effects are caused by damage to rapidly dividing normal cells in the area being treated. These effects include skin irritation or damage at regions exposed to the radiation beams. Examples include damage to the salivary glands or hair loss when the head or neck area is treated, or urinary problems when the lower abdomen is treated. Most acute effects disappear after treatment ends, though some (like

salivary gland damage) can be permanent. The drug amifostine (Ethyol®) can help protect the salivary glands from radiation damage if it is given during treatment. This type of drug is called a radioprotector. Fatigue is a common side effect of radiation therapy regardless of which part of the body is treated. Nausea with or without vomiting is common when the abdomen is treated and occurs sometimes when the brain is treated. Medications are available to help prevent or treat nausea and vomiting during treatment. Late side effects of radiation therapy may or may not occur. Depending on the area of the body treated, late side effects can include:

- Fibrosis (the replacement of normal tissue with scar tissue, leading to restricted movement of the affected area).
- Damage to the bowels, causing diarrhea and bleeding.
- Memory loss.
- Fertility (inability to have a child).
- Rarely, a second cancer caused by radiation exposure.

The link between radiation and cancer was confirmed many years ago through studies of the survivors of the atomic bombs in Japan, the exposures of workers in certain jobs, and patients treated with radiation therapy for cancer and other diseases. Some cases of leukemia are related to past radiation exposure. Most develop within a few years of exposure, with the risk peaking at 5 to 9 years, and then slowly declining. Other types of cancer that develop after radiation exposure have been found to take much longer to show up. These are solid tumor cancers, like cancer of the breast or lung. Most are not seen for at least 10 years after radiation exposure, and some are diagnosed even more than 15 years later. Second cancers that develop after radiation therapy depend on the part of the body that was treated. For example, girls treated with radiation to the chest for Hodgkin lymphoma have an increased risk of developing breast cancer later in life. In general, the lifetime risk of a second cancer is highest in people treated for cancer as children or adolescents. Whether or not a patient experiences late side effects depends on other aspects of their cancer treatment in addition to radiation therapy, as well as their individual risk factors. Some chemotherapy drugs, genetic risk factors, and lifestyle factors (such as smoking) can also increase the risk of late side effects. When suggesting radiation therapy as part of a patient's cancer treatment, the radiation oncologist will carefully weigh the known risks of treatment against the potential benefits for each patient (including relief of symptoms, shrinking a tumor, or potential cure). Radiation therapy techniques have steadily improved over the last few decades. Treatments now target the cancers more precisely, and more is known about setting radiation doses. These advances are expected to reduce the number of secondary cancers that result from radiation therapy. Overall, the risk of second cancers is low and must be weighed against the benefits gained with radiation treatments.

Nausea and vomiting : Nausea and vomiting are most likely to occur when the radiation dose is high or if the abdomen or another part of the digestive tract is irradiated. Sometimes

nausea and vomiting occur after radiation to other regions, but in these cases the symptoms usually disappear within a few hours after treatment. Nausea and vomiting can be treated with antacids, Compazine, Tigan, or Zofran.

Fatigue : Fatigue is an extreme tiredness that does not get better with rest. It's a common effect of radiation, but the exact cause is unknown. Fatigue frequently starts after the second week of therapy and may continue until about two weeks after the therapy is finished. Patients may need to limit their activities, take naps, and get extra sleep at night. Patients should see their oncologist at least once within the first few weeks after their final radiation treatment. They should also see an oncologist every six to twelve months for the rest of their lives so they can be checked to see if the tumor has reappeared or spread. Sometimes tumors cause the immune system to make substances that lead to fatigue. Fatigue may also be caused by anemia (a low red blood cell count), poor nutrition, pain, certain drugs such as steroids or chemotherapy, depression, and stress. There's no single treatment for fatigue, but if a cause can be found it should be treated. For example, if the fatigue is caused by anemia, some patients may benefit from blood transfusions or from medicines that cause the body to make more red blood cells. Fatigue can last for a long time after treatment is over and some people never have as much energy as they did before treatment. Light or moderate exercise with frequent rest breaks may help to reduce fatigue.

Damage to the epithelial surfaces : Radiation therapy may damage epithelial surfaces this may include the skin, oral mucosa, pharyngeal, bowel mucosa and ureter depending on the area being treated. The rates of damage and recovery from radiation therapy depend upon the turnover rate of epithelial cells. Typically during the treatment period the skin starts to become pink and sore several weeks. The reaction may become more severe during the treatment and for up to about one week following the end of radiation therapy, and the skin may break down. Although this moist desquamation is uncomfortable, recovery is usually quick. Skin reactions tend to be worse in areas where there are natural folds in the skin, such as underneath the female breast, behind the ear, and in the groin.

Mouth and throat problems : Mucositis (inflammation inside the mouth and throat) is a short-term side effect that can happen when radiation is given to the head and neck area. It can make swallowing painful, and some patients lose weight because they have trouble eating. It usually gets better within a few weeks after treatments end. Dry mouth and a loss of taste can be caused by radiation damage to the salivary glands and taste buds. Thick, sticky, rope-like saliva and swallowing problems may develop, too. These side effects often go away after treatments end, but sometimes they don't. Good nutrition is important for people with cancer. If mouth pain and irritation make it hard to eat or swallow, we may need to have a feeding tube put into our stomach for a while so we can take in enough nourishment. Radiation to the head and neck area can affect teeth, too, and increase chances of getting cavities. Mouth care to prevent problems will be an important part of treatment.

Dryness : The salivary glands and tear glands have a radiation tolerance of about 30 Gy

in 2 Gy fractions, a dose which is exceeded by most radical head and neck cancer treatments. Dry mouth (xerostomia) and dry eyes (xerophthalmia) can become irritating long-term problems and severely reduce the patient's quality of life. Similarly, sweat glands in treated skin (such as the armpit) tend to stop working, and the naturally moist vaginal mucosa is often dry following pelvic irradiation.

Lymphedema : Lymphedema, a condition of localized fluid retention and tissue swelling, can result from damage to the lymphatic system sustained during radiation therapy. It is the most commonly reported complication in breast radiation therapy patients who receive adjuvant axillary radiotherapy following surgery to clear the axillary lymph nodes

Hair loss : Radiation therapy can cause hair loss but hair is only lost in the area being treated. For instance, radiation to our head may cause lose some or all of the hair on our head (even eyebrows and lashes), but if we get treatment to our hip, we will not lose the hair on our head. Most people find that their hair grows back after treatment ends, but it can be hard to deal with hair loss. When it does grow back, our hair may be thinner or a different texture than it was before. If we lose our hair, our scalp may be tender and we may want to cover our head. Wear a hat or scarf to protect our head when we are in the sun.

Blood count changes : Radiation therapy can cause low white blood cell counts or low levels of platelets, but this is rare. If our blood tests show changes in our counts, treatment might be delayed for a week or so to allow our blood counts to return to normal.

Joint or muscle stiffness : Occasionally people who have radiotherapy to areas over joints or muscles may experience some stiffness. This can occur at any time up to two years after treatment has finished. Regular exercise to these joints and muscles can prevent stiffness.

Intestinal discomfort: The lower bowel may be treated directly with radiation (treatment of rectal or anal cancer) or be exposed by radiation therapy to other pelvic structures (prostate, bladder, female genital tract). Typical symptoms are soreness, diarrhoea, and nausea.

Swelling (edema or oedema) : Swelling of soft tissues may cause problems during radiation therapy. This is a concern during treatment of brain tumors and brain metastases, especially where there is pre-existing raised intracranial pressure or where the tumor is causing near-total obstruction of a lumen (e.g., trachea or main bronchus). Surgical intervention may be considered prior to treatment with radiation. If surgery is deemed unnecessary or inappropriate, the patient may receive steroids during radiation therapy to reduce swelling.

Side effects of radiation to specific areas :

Brain : Radiation therapy to large areas of the brain can sometimes cause changes in brain function that can lead to memory loss, lower sexual desire, or poor tolerance for cold weather. Nausea, unsteady walking, and changes in vision may also be noticed. Usually these symptoms are minor compared to those caused by a brain tumor, but they can be troublesome. Sometimes a large area of dead cells, called radiation necrosis, forms at the site of the radiation in the brain. This can happen months to years after radiation is given, and can

cause symptoms like seizures, mental status changes, headaches, trouble speaking or walking, and other changes. It can be hard to tell if these symptoms are from necrosis or from the tumor coming back. Patients with radiation necrosis usually do better than patients whose brain tumors come back. But still, a number of patients with radiation necrosis do poorly or even die.

Lung : When radiation treatments include the chest, it can affect the lungs. One early change is a decrease in the levels of a substance called surfactant, which helps keep the air passages open. Low surfactant levels keep the lungs from fully expanding. This may cause shortness of breath or a cough. These symptoms are sometimes treated with steroids. Depending on the location of the area getting radiation, some people also have trouble swallowing. Radiation pneumonitis occurs in about 5 to 20% of people get radiation therapy for lung cancer. It can also be caused by radiation to the chest for breast cancer, lymphomas, or other cancers. This inflammation may occur from about 6 weeks to 6 months after completing external radiation therapy. Common symptoms include shortness of breath, chest pain, cough, and fever. Radiation pneumonitis is treated by trying to decrease the inflammation. Steroids, like prednisone, are usually used. Another possible effect radiation can have on the lungs is fibrosis (stiffening or scarring). This means the lungs are less able to expand and take in air. Fibrosis can cause shortness of breath and make it hard to exercise. This problem may show up months or even years after treatment.

Digestive tract : Radiation to the chest and abdomen (belly) may cause swelling and inflammation in the esophagus (the tube connecting the throat to the stomach), stomach, or intestine (bowels). This can cause pain, nausea, vomiting, or diarrhea. Antacids, sometimes combined with a numbing medicine such as lidocaine, may help relieve pain from an inflamed esophagus. Nausea and vomiting can also be treated with medicines. If it's severe, some patients may need intravenous (IV) fluids to avoid or treat dehydration and strong medicines like morphine to treat pain. Diarrhea also can be treated with medicines and may be helped by avoiding spicy, fried, or high fiber foods. Digestive problems usually go away within a week or 2 of the last radiation treatment. Rarely serious enough to cause long-term problems such as scarring that can cause permanent narrowing of the esophagus, or ulcers that can cause abnormal openings in the intestine. Diarrhea and bleeding can result if the colon or rectum is affected (colitis or proctitis).

Reproductive/sex organs : Fertility in men: Radiation to the testicles can cause permanent loss of sperm production. Unless the cancer is in the testicles, they can usually be protected from radiation by using a shield called a clam shell.

Fertility in women : It's harder to protect the ovaries when women are getting radiation to the abdomen (belly). If both ovaries are exposed to radiation, early menopause and permanent infertility can result. Sparing one ovary can prevent these side effects. If the uterus (womb) is exposed, radiation can cause scarring and fibrosis.

Sexual effects of radiation therapy on women : Radiation to the pelvic area can make

the vagina tender and inflamed during and for a few weeks after treatment. The area may scar as it heals. This scarring can interfere with the vagina's ability to stretch. The lining of the vagina also gets thinner, and might bleed slightly after sex. A few women get ulcers, or sore spots, in their vaginas. It may take many months for these areas to heal after radiation therapy ends. The scarring that normally occurs after pelvic radiation can also shorten or narrow the vagina so much that a woman might not be able to have sex or get a Pap smear without pain. This can often be prevented by stretching the walls of the vagina a few times a week. One way to do this is to have sex that includes vaginal penetration at least 3 to 4 times a week. Another option is to use a vaginal dilator. A dilator is a plastic or rubber rod or tube used to stretch out the vagina. It feels much like putting in a large tampon for a few minutes. Even if a woman is not interested in staying sexually active, it helps allow her doctor to do pelvic exams. This is an important part of follow-up care after treatment. As long as a woman is not bleeding heavily from a tumor in her bladder, rectum, uterus, cervix, or vagina, she may be able to have sex during pelvic radiation therapy. The outer genitals and vagina are just as sensitive as usual. But if any of these areas are being radiated, sex may be uncomfortable because of sore spots or inflamed tissues in the vulva or vagina. Other side effects of radiation can also make a person less interested in sex during treatment.

Sexual impact of radiation therapy on men : Radiation therapy to the pelvis can damage the arteries and nerves that supply the penis and as a result, cause problems with erections. The higher the dose of radiation and the wider the area of the pelvis treated, the greater the chance that a man will develop erection problems. Men who are older, who didn't have full erections before they were treated, who have high blood pressure, or who have been heavy smokers seem to have a higher risk of having erection problems after radiation. Testosterone is a male hormone that plays an important role in erections. Some men have less testosterone after pelvic radiation. The testicles, which make testosterone, may be affected either by a mild dose of scattered radiation or by the general stress of cancer treatment. Testosterone levels usually return to normal within 6 months of radiation therapy. But if a man has problems with low sexual desire after cancer treatment, the doctor may decide to do a blood test to find out if testosterone is low. Some men can take testosterone to raise low levels to normal. Men with prostate cancer should know that replacement testosterone can speed up the growth of prostate cancer cells.

New radiation therapy : New ways of delivering radiation therapy are making it safer and more effective. Some of these methods are already being used, while others need more study before they can be approved for widespread use. Scientists around the world continue to look for better and different ways to use radiation to treat cancer. Here are just a few areas of current research interest:

Hyperthermia is the use of heat to treat cancer. Heat has been found to kill cancer cells, but when used alone it does not destroy enough cells to cure the cancer. Heat created by microwaves and ultrasound is being studied in combination with radiation and appears to

improve the effect of the radiation.

Hyperbaric oxygen therapy consists of breathing pure oxygen while in a sealed chamber that has been pressurized at 1½ to 3 times normal atmospheric pressure. It helps to increase the sensitivity of certain cancer types to radiation. It's also being tested to see if it can reverse some of the damage to normal body tissues caused by radiation.

Radiosensitizers are a growing field in cancer treatment. Researchers are continuing to look for new substances that will make tumors more sensitive to radiation without affecting normal tissues.

Radioprotectors are substances that protect normal cells from radiation. These types of drugs are useful in areas where it's hard not to expose vital normal tissues to radiation when treating a tumor, such as the head and neck area. Some radioprotectors, such as amifostine (Ethyol®), are already in use, while others are being studied in clinical trials.

Research is being done to improve radiation therapy:

Clinical trials are conducting by doctors and other scientists to know the use of radiation therapy for cancer more safely and effectively. Clinical trials allow researchers to examine the effectiveness of new treatments in comparison with standard ones, as well as to compare the side effects of the treatments. Researchers are working on improving image-guided radiation so that it provides real-time imaging of the tumor target during treatment. Real-time imaging could help compensate for normal movement of the internal organs from breathing and for changes in tumor size during treatment. Researchers are also studying radio sensitizers and radio protectors, chemicals that modify a cell's response to radiation. Radio sensitizers are drugs that make cancer cells more sensitive to the effects of radiation therapy. Several agents are under study as radio sensitizers. In addition, some anticancer drugs, such as 5-fluorouracil and cisplatin, make cancer cells more sensitive to radiation therapy. Radio protectors (also called radio protectants) are drugs that protect normal cells from damage caused by radiation therapy. These drugs promote the repair of normal cells exposed to radiation. Many agents are currently being studied as potential radio protectors. The use of carbon ion beams in radiation therapy is being investigated by researchers, but, at this time, the use of these beams remains experimental. Carbon ion beams are available at only a few medical centers around the world. Researchers hope that carbon ion beams may be effective in treating some tumors that are resistant to traditional radiation therapy.

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CHEMOTHERAPY

The term chemotherapy was coined by Paul Ehrlich in the early 1900s as meaning use of any chemicals to treat any disease (chemo- + -therapy), such as the use of antibiotics (antibacterial chemotherapy). The first modern chemotherapeutic agent was arsphenamine, an arsenic compound used to treat syphilis discovered in 1909. This was later followed by sulfonamides (sulfa drugs) and penicillin. In today's usage, the sense "any treatment of disease with drugs" is often expressed with the word pharmacotherapy.

Chemotherapy (abbreviated to chemo and sometimes CTX or CTx) is the use of medication (chemicals) to treat disease specifically destruction of cancer cells by interfere with the process of cell division. They do this either by damaging the proteins involved, or by damaging the DNA itself. This causes the damaged cells to commit suicide (by apoptosis). However, chemotherapy may also include the use of antibiotics or other medications to treat any illness or infection. Chemotherapy may be given with a curative intent, prolong life span or to reduce symptoms. Along with hormonal therapy and targeted therapy, it is one of the major categories of medical oncology (pharmacotherapy for cancer). These modalities are often used in conjunction with other cancer treatments, such as radiation therapy, surgery, and/or hyperthermia therapy. Chemotherapy may use single-agent chemotherapy (one drug at a time) or combination chemotherapy or polychemotherapy (several drugs at a time). The combination of chemotherapy and radiotherapy is commonly known as chemoradiotherapy. Chemotherapy using drugs that convert to cytotoxic activity after light exposure is called photochemotherapy or photodynamic therapy. Chemotherapeutic drugs are effective against cancer cells because cancerous cells divide rapidly, whereas most normal cells in our body do not. However, some cells in our body, such as bone marrow cells, immune cells and hair follicle cells divide rapidly and are often damaged during chemotherapy. As a result, chemotherapy drugs often have unpleasant side effects such as nausea, hair loss and vomiting.

History of cancer chemotherapy

The beginnings of the modern era of cancer chemotherapy can be traced directly to the German introduction of chemical warfare during World War I. Among the chemical agents used, mustard gas was particularly devastating. In 1925, mustard gas was banned by the Geneva Protocol; the advent of World War II caused concerns over the possible re-introduction of chemical warfare. These concerns led to the discovery of nitrogen mustard, a chemical warfare agent, as an effective treatment for cancer. Department of Defense in the United States was recruited two pharmacologists, Louis S. Goodman and Alfred Gilman, to investigate potential therapeutic applications of chemical warfare agents. They observed that mustard gas was suitable for laboratory experiments because of its too volatile nature. They exchanged a nitrogen molecule for sulfur and had a more stable compound in nitrogen mustard. A year into the start of their research a German air raid in Bari, Italy led to the

exposure of more than one thousand people to the SS John Harvey's secret cargo composed of mustard gas bombs. Dr. Stewart Francis Alexander, a Lieutenant Colonel who was an expert in chemical warfare, was subsequently deployed to investigate the aftermath. Autopsies of the victims suggested that profound lymphoid and myeloid suppression had occurred after exposure. In his report Dr. Alexander theorized that since mustard gas all but ceased the division of certain types of somatic cells whose nature it was to divide fast, it could also potentially be put to use in helping to suppress the division of certain types of cancerous cells.

Using this information, Goodman and Gilman reasoned that this agent could be used to treat lymphoma, since lymphoma is a tumor of lymphoid cells. They first set up an animal model — they established lymphomas in mice and demonstrated they could treat them with mustard agents. Next, in collaboration with a thoracic surgeon, Gustaf Lindskog, they injected a related agent, mustine (prototype nitrogen mustard), into a patient with non-Hodgkin's lymphoma. They observed a dramatic reduction in the patient's tumour masses. Although this effect lasted only a few weeks, and the patient had to return for another set of treatment, this was the first step to the realization that cancer could be treated by pharmacological agents.

Shortly after World War II, a second approach to drug therapy of cancer began. Sidney Farber, a pathologist at Harvard Medical School, studied the effects of folic acid on leukemia patients. In 1937, Lucy Wills, when working in India, discovered the crucial role of Folic acid (a vitamin), in DNA metabolism (he did not know the significance of DNA at that time). It seemed to stimulate the proliferation of acute lymphoblastic leukemia (ALL) cells when administered to children with this cancer. In one of the first examples of rational drug design (rather than accidental discovery), in collaboration with Harriett Kilde and Yellapragada Subbarow of Lederle Laboratories, Farber used folate analogues. These analogues—first aminopterin and then amethopterin (now methotrexate) were antagonistic to folic acid, and blocked the function of folate-requiring enzymes. When administered to children with ALL in 1948, these agents became the first drugs to induce remission in children with ALL. Remissions were brief, but the principle was clear—antifolates could suppress proliferation of malignant cells, and could thereby re-establish normal bone-marrow function. Farber met resistance to conducting his studies at a time when the commonly held medical belief was that leukemia was incurable, and that the children should be allowed to die in peace. Afterwards, Farber's 1948 report in the *New England Journal of Medicine* was met with incredulity and ridicule.

Nitrogen mustard works by physically modifying the DNA in our cells - it is a class of drug called an 'alkylating agent'. In the 1950s in London, Cancer Research UK (then the Cancer Research Campaign) led to the development of more effective alkylating agents, chlorambucil, melphalan and busulphan. These drugs are still regarded as mainstays of therapy for myeloma, lymphoma and some leukaemias. Another class of chemotherapy drug was discovered in the 1950s - the 'antimetabolites'. These drugs resemble natural chemicals in the body, but act to block up the proteins that normally deal with them. Antimetabolites

such as 6-mercaptopurine and 5-fluorouracil are still in widespread use today, often as part of combination therapy.

In 1951, Jane C. Wright demonstrated the use of methotrexate in solid tumors, showing remission in breast cancer. Wright's group was the first to demonstrate use of the drug in solid tumors, as opposed to leukemias, which are a cancer of the marrow. Several years later at the National Cancer Institute, Roy Hertz and Min Chiu Li then demonstrated complete remission in women with choriocarcinoma and chorioadenoma in 1956, discovering that methotrexate alone could cure choriocarcinoma (1958), a germ-cell malignancy that originates in trophoblastic cells of the placenta.

The platinum derivatives make up another important class of chemotherapy drug. These were first discovered in 1965, when Barnett Rosenberg in Michigan was studying whether bacteria can grow in electric fields. In his experiments, he used platinum electrodes, because platinum does not usually react with other substances. He noticed that the bacteria stopped dividing when the electric field was turned on, and assumed that the electric field had stopped them dividing. However, when he turned the field off, rather than starting to divide again, the bacteria did not resume cell division. This meant that something else had caused them to stop dividing. After further work, he discovered that the platinum electrode had unexpectedly reacted with the liquid where the bacteria were growing and form a highly toxic compound. Many years later, thanks largely to pioneering work by Cancer Research UK's Tom Connors, the first platinum derivative, cisplatin, was approved for use in cancer patients. In 1977, cisplatin, in combination with other drugs, had revolutionized the treatment of testicular cancer, and significantly improved the treatment of many other cancers. In some ways, chemotherapy is rather like taking a sledgehammer to crack a hazelnut. The drugs will affect normal body tissues as well as cancer cells that cause the side effects. Cancer research is committed to producing a new generation of more specific, better-targeted cancer therapies, to improve the quality of life of cancer patients and to minimize the risks from their treatment.

Chemotherapy has five possible goals

- Total remission - to cure the patient completely. In some cases only chemotherapy can get rid of the cancer completely.
- Combination therapy-chemotherapy can help other therapies, such as radiotherapy or surgery.
- Delay/Prevent recurrence - chemotherapy, when used to prevent the return of a cancer, is most often used after a tumor is removed surgically. Scientists at the Charite School of Medicine, Germany, found that the use of the drug gemcitabine for chemotherapy significantly delays the recurrence of cancer, compared to no chemotherapy.
- Slow down cancer progression - used mainly when the cancer is in its advanced stages and a cure is unlikely. Chemotherapy can slow down the advancement of the cancer.
- To relieve symptoms - more frequently used for patients with advanced cancer.

Types of Chemotherapy Drugs:

Paul Ehrlich originated the term chemotherapy for searching a substance to cure syphilis in 1914. Although many agents have been developed to treat infectious diseases induced by bacteria, the development of anti-cancer agents has been done by trial and error, serendipity or empiricism methods. Depending on the type of cancer and the kind of drug used, chemotherapy drugs may be administered differently such as orally (oral chemotherapy), or injected into a muscle (intramuscular injection), injected under the skin (subcutaneous injection), or into a vein (intravenous chemotherapy). Intravenous chemotherapy was the most commonly used method for administration of chemotherapeutic drugs. Medication in to the blood stream by intravenous injection was the most efficient way. In special cases, chemotherapy drugs may be injected into the fluid around the spine (intrathecal chemotherapy). Two or more methods of administration may be used at the same time under certain circumstances. No matter what method is used, chemotherapy drugs are absorbed into the blood and carried around the body. Oral chemotherapy is more convenient and does not require any specialized equipment. In chemotherapy, one or more available anti-cancer drugs may be given to cancer patients. Different phases in the cell cycle, different chemical agent's damage cancer cells in different ways. To increase the cancerous cell killing effectively a combination of drug is employed which is called combination chemotherapy. These agents are classified according to their composite structure, similarity to other compounds, derivation, and mechanism of intracellular action. Engineered anti-cancer agents (designer drugs) such as monoclonal antibodies, cytokines, gene therapy vectors, and antisense and peptide molecules have only been developed recently. Specific therapies are dependent on metabolic functions, which differ from normal cells, and are unique to the malignant cell. Fundamental experimental observations support the concept that chemotherapeutic agents primarily function by inhibiting mitosis (cell reproduction) and inducing apoptosis (cell death). The different types of chemotherapy drugs includes

Alkylating agents : Alkylating agents were among the first anti-cancer a drug beginning in the 1940's and are the most commonly used in chemotherapy today. Alkylating agents are most active in the resting phase of the cell. These types of drugs are cell-cycle non-specific meaning that they kill the cell in various and multiple phases of the cell cycle. While there are many different classes of alkylating agents, they all work by this same chemical mechanism. Alkylating drugs have this effect on a cancer cell during every phase of its life cycle, making them desirable for use on a wide range of cancers. The most benefit is seen in their use to treat cancers that grow slowly, like solid tumors and leukemia, but they are also used to treat lung cancer, ovarian cancer, breast cancer, lymphomas, sarcomas, myelomas, and Hodgkin's disease. Alkylating agents are not as effective on rapidly growing cells. The biggest weakness of most cancer cells is that they are very sensitive to DNA damage. Alkylating agents work by reacting with the proteins that bond together to form the very delicate double helix structure of a DNA molecule, adding an alkyl group to some or all of them. This prevents the proteins from linking up as they should, cause breakage of the DNA strands and, eventually, the death

of the cancer cell. This phenomenon is essentially a mutation that takes away the cancer cell's ability to multiply. The five major categories of alkylating agents are nitrogen mustards, nitrosoureas, alkyl sulfonates, triazines, and ethylenimines. In all cases, dosage and schedule are determined on an individual basis, considering the patient's size, overall health, and the type of cancer being treated. There are several types of alkylating agents used in chemotherapy treatments namely:

- Mustard gas derivatives: Mechlorethamine, Cyclophosphamide, Chlorambucil, Melphalan, Ifosfamide, Trofosfamide and Estramustine.
- Ethylenimines: Thiotepa and Hexamethylmelamine, Triaziquone and Carboquone.
- Alkylsulfonates: Busulfan.
- Hydrazines and Triazines: Altretamine, Procarbazine, Dacarbazine and Temozolomide.
- Nitrosoureas: Carmustine, Lomustine and Streptozocin. Nitrosoureas are unique because, unlike most types of chemo treatments, they can cross the blood-brain barrier. They can be useful in treating brain tumors.
- Metal salts: Carboplatin, Cisplatin, and Oxaliplatin.

Mustard gas derivatives:			
Generic name	Brand name	Use in cancer	Route
Mechlorethamine	Mustargen	Hodgkin disease, lymphosarcoma, certain leukemias, mycosis fungoides, and other cancers	IV
Cyclophosphamide	Cytosan, Neosar	Lymphoma, leukemia, and many carcinomas	Oral (tablet, liquid), IV
Chlorambucil	Leukeran	CLL, Hodgkin and non-Hodgkin lymphoma, others	Oral
Melphalan		Multiple myeloma, as well as ovarian, breast, and prostate cancer	Oral, IV
Ifosfamide	Ifex	Lymphoma, leukemia, testicular, and bladder cancers.	IV
Trofosfamide	Ixoten	Cyclophosphamide prodrug	
Estramustine	Emcyt	Advanced prostate cancer, conjugated with estradiol	Oral

Ethylenimines derivatives :			
Thiotepa	Thioplex Girostan Thiofozil Tifosyl	Lymphoma and cancers of the breast, ovary, and bladder	IV, Intrabladder, Intraperitoneal

Altretamine	Hexalen Hexamethyl melamine	Treatment-resistant ovarian cancer	Oral
Triaziquone		Not used clinically	
Carboquone		Not used clinically	

Alkylsulfonates

Busulfan	Myleran	CML, other blood cancers	Oral
Treosulfan	Ovastat (foreign)	Busulfan analogue	
Mannosulfan		Busulfan analogue	

Hydrazines and Triazines

Dacarbazine	DTIC-Dome	Hodgkin's lymphoma, malignant melanoma	IV
Temozolomide	Temodar	Cancers of brain including anaplastic astrocytoma, glioblastoma multiforme (GBM), and gliomas.	Oral, IV

Nitrosureas

Carmustine	Becenum BCNU Carmubris Gliadel	Lymphoma, multiple myeloma, gliomas, and other types of cancer.	IV Implantable wafer (glioma)
Lomustine	CCNU	Cancers of brain, Hodgkin lymphoma	Oral
Semustine	Methyl-CCNU	Related to carmustine	
Streptozocin	Zanosar		IV
Fotemustine	Muphoran (foreign)		
Nimustine	ACNU Nidran		
Ranimustine	Cymer (foreign) Cymerin (foreign)		

The action of alkylating agents as chemotherapeutic drugs comes from their ability to bind DNA irreversibly. Once bound, the altered molecule disrupts the normal action and replication of the DNA strand. When this alkylation takes place at several places along the DNA molecule, the normal processes of the cell are interrupted. This leads to either to programmed cell death (apoptosis) or at least an arrest in cellular replication. In either case, when applied to cancer cells, alkylating agents can limit tumor growth and lead to tumor

destruction. Neoplasms, especially consisting of certain cell types, are known to develop resistance to alkylating agents. This resistance has been linked, at least in part, to the expression of an enzyme known as O6-MethylguanineDNAmethyltransferase (MGMT). MGMT is able to repair DNA errors caused by alkylating agents. For example, temozolomide causes a potentially cytotoxic lesion in oxygen 6 of guanine nucleotides in DNA. MGMT enzymatically removes this methyl group, repairs the DNA, and negates the effect of the chemotherapy. In normal cells this would be advantageous; a cellular mechanism to prevent DNA disruption in cells that are normal physiologically. However cancers are also able to express this protein thus rendering certain alkylating agents ineffective. Drugs that inhibit MGMT activity may be used as an adjunct to alkylating agents in order to overcome this resistance and improve the tumor-killing effect.

Alkylating agents can cause long-term damage to the bone marrow due to DNA damage. In rare cases, this can eventually lead to acute leukemia. The risk of leukemia from alkylating agents is “dose-dependent,” meaning that the risk is small with lower doses, but goes up higher amount of the drug used. The risk of leukemia after getting alkylating agents is highest about 5 to 10 years after treatment. The platinum drugs (cisplatin, carboplatin, and oxaloplatin) are sometimes grouped with alkylating agents because they kill cells in a similar way. These drugs are less likely than the alkylating agents to cause leukemia later on. Occasionally severe organ damage can occur with the administration of alkylating agents. Pulmonary fibrosis and venoocclusive disease of the liver has occurred across all types of drugs within the class. The use of nitrosureas has been associated with renal failure. The central nervous system can be affected by alkylating agents as well. In addition to severe nausea and vomiting common to the class, certain agents (e.g. Ifosfamide) is quite neurotoxic, leading to acute confusion and delirium, seizures, paralysis, and coma. Alopecia (hair loss) is known to occur with alkylating agents. Sex organs are not spared—women that are treated with alkylating agents may experience permanent amenorrhea (lack of menstruation) and in men, sperm production may cease.

Plant Alkaloids : The vinca alkaloids are a subset of drugs derived from the Madagascar periwinkle plant. They were discovered in the 1950’s by Canadian scientists, Robert Noble and Charles Beer. The vinca alkaloids are made from the periwinkle plant (*catharanthus rosea*). The taxanes are made from the bark of the Pacific Yew tree (*taxus*). The vinca alkaloids and taxanes are also known as antimicrotubule agents. The podophyllotoxins are derived from the May apple plant. Camptothecan analogs are derived from the Asian “Happy Tree” (*Camptotheca acuminata*). Podophyllotoxins and camptothecan analogs are also known as topoisomerase inhibitors, which are used in certain types of chemotherapy. The plant alkaloids are cell-cycle specific means they attack the cells during various phases of division. Different types of alkaloids are

Vinca alkaloids : Vincristine, Vinblastine and Vinorelbine.

Taxanes : Paclitaxel and Docetaxel.

Podophyllotoxins : Etoposide and Teniposide.

Camptothecan analogs : Irinotecan and Topotecan.

Vinca alkaloids have been used to treat diabetes, high blood pressure, and the drugs have even been used as disinfectants. However, the vinca alkaloids are most famous for being cancer fighters. There are four major vinca alkaloids in clinical use namely vinblastine, vinorelbine, vincristine, and vindesine. Different vinca alkaloids have their own unique properties. All vinca alkaloids are administered intravenously (IV), eventually metabolized by the liver and excreted. The vinca alkaloids are cytotoxic means they halt the division of cells and cause cell death. During cell division, vinca alkaloid molecules bind to the building blocks of a protein called tubulin, inhibiting its formation. Tubulin protein normally works in cells to create “spindle fibers,” (also called microtubules). These microtubules provide cells with both the structure and flexibility they need to divide and replicate. Without microtubules, cells cannot divide. The vinca alkaloid’s mechanism in a nutshell by occupying tubulin’s building block structure, prevent cancer cells from successfully dividing. Vinblastine inhibits angiogenesis or the process by which new blood vessels grow from pre-existing ones. Angiogenesis is an essential step in a tumor’s transition to malignancy. Vinblastine is most often applied to treat Hodgkin’s disease, non-Hodgkin’s lymphoma, breast cancer, and germ cell tumors. Vinblastine caused several Side effects includes toxicity to white blood cells, nausea, vomiting, constipation, dyspnea, chest or tumor pain, wheezing, and fever. Vinblastine is also occasionally associated with antidiuretic hormone secretion and angina. Vinorelbine acts the same way as vinblastine. Vinorelbine has exhibited significant antitumor activity in patients with breast cancer and antiproliferation effects on osteosarcoma (bone tumor cells). Whereas it has been decrease the stability of lipid bilayer membranes (like those of a cell’s). Vinorelbine’s side effect includes decreased resistance to infection, bruising or bleeding, anemia, constipation, diarrhea, nausea, numbness or tingling in the hands and feet, fatigue (peripheral neuropathy) and inflammation at the injection site. Less common side effects include hair loss and allergic reaction. Vincristine’s inhibition of microtubule formation is especially powerful. The reason for this comes from the fact that tubulin protein is dynamic. Its long chain of building blocks is always growing in some places and breaking in others. The less contiguous parts of a tubulin molecule have pieces only two building blocks long, called dimers. Vincristine has a high affinity for tubulin dimers. The reaction between vincristine and the dimers is rapidly reversible means a vincristine molecule will attach to a dimer at one site, break off, and then reattach at another site. This keeps two sites per dimer “poisoned” and unable to reassemble into the protein. So vincristine’s ability to destabilize tubulin is especially good. FDA approved Vincristine to treat acute leukemia, rhabdomyosarcoma, neuroblastoma, Wilm’s tumor, Hodgkin’s disease, and other lymphomas. The most common side effects of Vincristine’s are peripheral neuropathy, suppression of bone marrow activity, constipation, nervous system toxicity, nausea, and vomiting. Vindesine is administered at a dose of 3 milligrams per square meter of body surface. The drug is applied to treat melanoma,

lung cancers, and (combined with other drugs) uterine cancers. Additional side effects from vindesine include anemia, blood cell toxicity, fatigue, tingling or pricking sensations in the skin, and skin toxicity. Overall, vinca alkaloids are in the second most-used class of cancer drugs but sales are expected to decline. Nonetheless, vinca alkaloids will remain among the fundamental cancer therapies. Research for new vinca alkaloid applications is ongoing.

Antimetabolites : Antimetabolites are drugs that interfere with one or more enzymes or their reactions that are necessary for DNA synthesis. They affect DNA synthesis by acting as a substitute to the actual metabolites that would be used in the normal metabolism (for example antifolates interfere with the use of folic acid).

Antimetabolites are drugs used in cancer chemotherapy. Cancer cells divide more rapidly compared to normal cells so antimetabolites affect cancer cell replication more than they affect normal cell replication. Antimetabolites are cell-cycle specific. They attack cells at very specific phases in the cycle. Antimetabolites are classified according to the substances with which they interfere.

Folic acid antagonist : Methotrexate.

Pyrimidine antagonist : 5-Fluorouracil, Foxuridine, Cytarabine, Capecitabine, and Gemcitabine.

Purine antagonist : 6-Mercaptopurine and 6-Thioguanine.

Adenosine deaminase inhibitor : Folic acid, pyrimidine or purine analogues are the most effective antimetabolites chemotherapeutic drugs which are characterized by low molecular weights. They have similar structures as naturally occurring molecules used in nucleic acid (DNA and RNA) synthesis. Antimetabolites are similar to chemicals needed for normal biochemical activity, but differ enough so that they interfere with normal cell function. Generally, antimetabolites induce cell death during the S phase of cell growth when incorporated into RNA and DNA or inhibit enzymes needed for nucleic acid production. These agents are used for a variety of cancer therapies including leukemia, breast, ovarian and gastro-intestinal cancers. Some examples of antimetabolite are Cladribine, Fludarabine, Nelarabine and Pentostatin.

Folate antagonist class : Folic acid is a necessary element for the production of nucleotides. It was empirically observed in patients with leukemia, that diets low in folate produced lower white cell counts as compared to leukemic patients on normal folate diets. In 1948, a folate antagonist methotrexate was developed which is found effective in childhood leukemia. Methotrexate had less toxic side effects and clinically more effective. Intracellularly, folate is converted by the enzyme dihydrofolate reductase (DHFR) to dihydrofolate then reduced to active folate commonly known as tetrahydrofolate (THDF). THDF acts as a carbon carrier compound that donates methyl groups to end target molecules through the enzymatic action of thymidine synthetase (TS). DHFR is continuously used in this process and is the site where the folate antagonists function. These drugs generally function by impeding enzyme action. Methotrexate binds to the enzyme DHFR reversibly and inactivates it. This

prevents methylation and decreases available supplies of purine and thymidine bases for new DNA and RNA synthesis. It is active in the S phase of cell cycle. Methotrexate remains the primary folate antagonist used today even though others have been discovered. It is effective in many malignancies namely breast, head and neck, colorectal, non-Hodgkin's lymphomas, osteosarcoma, bladder and choriocarcinoma. It is also used in acute lymphocytic leukemia, and some types of meningeal carcinomas. The primary complication of methotrexate treatment was drug resistance and decreased drug transport into the cell can occur. A lower and less effective dose of methotrexate is observed intracellularly. Genetic mutations and alterations in gene activity may occur as well which alter binding constants to the enzymes or increases in the DHFR enzyme within the cell. Resistance is a major contributor to treatment failure with this drug. Other folate antagonist pemetrexed combined with cisplatin is effective in the treatment of mesothelioma and non-small cell lung cancer. Pemetrexed acts like methotrexate. It hinders multiple enzymes needed for de novo production of the thymidine and purine nucleotides. Normal DNA and RNA production is prevented.

Pyrimidine compounds : Duschinsky synthesized and Heidelberger, in 1957, introduced 5-fluorouracil (5-FU), a pyrimidine base containing a fluoride atom at the 5 carbon position on the ring. Uracil is a naturally occurring pyrimidine base used in nucleic acid synthesis. It is converted to thymidine by adding a methyl group at the fifth carbon of the pyrimidine ring by enzyme thymidylate synthetase. 5-FU is similar in structure to uracil and is converted to two active metabolites (FdUMP and FUTP) that inhibit the activity of the enzyme thymidylate synthetase. 5-FU mimics the natural base and functions to inhibit DNA synthesis. The methyl group cannot be added because of the fluoride atom at the five positions as a result normal DNA synthesis fails. dUTP and FdUTP are incorporated into DNA so that it cannot function normally. In addition, FUTP is incorporated into RNA leading to faulty translation of the RNA. Thus, the synthesis of multiple forms of RNA (messenger, ribosomal, transfer and small nuclear RNAs) is blocked. These combined actions on DNA and RNA are cytotoxic to the rapidly dividing cancer cells. 5-FU is used for the treatment of many malignancies such as breast, head and neck, adrenal, pancreatic, gastric, colon, rectal, esophageal, liver and G-U (bladder, penile, vulva, prostate). 5-FU may be administered by bolus IV infusion or continuous IV infusion over two days every 2-3 weeks or by oral ingestion. In addition, it may be used to treat skin cancers (basal cell and keratosis) by topical application. Other pyrimidine antagonists are arabinosylcytosine, capecitabine, gemcitabine and decitabine. Arabinosylcytosine or cytarabine is a deoxycytidine base compound that is converted to its active metabolite, ara-CTP. This base is incorporated into DNA and causes strand termination as a result the cancer cell is unable to divide. It is effective in acute non-lymphocytic, lymphocytic, myelogenous and chronic myelocytic leukemias, as well as leptomeningeal carcinomatosis and non-Hodgkin's lymphoma. Capecitabine is an oral 5-FU pro-drug and converted to 5-FU by actions in liver and tumor cells. It is used as adjuvant therapy in colon and breast metastasis. Gemcitabine is an ara-C pro drug which is activated by intracellular phosphorylation. This inhibits DNA and RNA synthesis. It is a first line treatment

of pancreatic, metastatic breast, bladder, ovarian and non-small cell lung cancers. Finally, decitabine is phosphorylated and directly incorporated into DNA. In cancer cells, it stops methylation by inhibiting DNA methyltransferase and induces cell death. It may also restore normal gene function controlling cell proliferation. It is used therapeutically in myeloplastic syndrome.

Purine compounds : The pyrimidine bases (uracil, cytosine), and the purine bases (adenine, guanine) are building blocks in the synthesis of DNA and RNA nucleotides. In the replication process, nucleotides are joined to one another to form DNA strands. It is less clear how the purine antagonists function, but they may inhibit normal production of DNA. It is conjectured that these purine antagonists stop synthesis by decreasing the production of the purine bases or may be incorporated into the DNA strands during synthesis and halt cell replication. Genetic mutation may lead to purine resistance. Fludarabine or 2-fluoro-ara-amp is an antimetabolite of the purine class. It functions as a pro-drug, dephosphorylated and enters the cancer cell. Fludarabine is rephosphorylated to F-ara-ATP and incorporation into the DNA strand, it halts strand lengthening. The drug is successfully used in treating refractory chronic lymphocytic and chronic B cell leukemias, non-Hodgkin's lymphoma and T- cell lymphoma. 6-Mercaptopurine (6-MP) is another purine agent successfully used against acute lymphocytic leukemia. It is active in the S phase of cell proliferation. Upon incorporation into DNA and RNA, the nucleic acids are rendered useless. 6-MP may also act through inhibition of de novo synthesis of the purine bases. Without adequate amounts of the purine bases, nucleotide production stops and the cancer cell dies.

Antitumor Antibiotics : Antitumor antibiotics are chemo treatments made from natural products produced by species of the soil fungus *Streptomyces*. These drugs act during multiple phases of the cell cycle and are considered cell-cycle specific. There are several types of antitumor antibiotics such as:

Anthracyclines : Doxorubicin, Daunorubicin, Epirubicin, Mitoxantrone, and Idarubicin.

Chromomycins : Dactinomycin and Plicamycin.

Miscellaneous : Mitomycin and Bleomycin.

Anthracyclines are anticancer compounds that were originally derived from *Streptomyces* and their anti-tumor activities were established in the 1960s. Anthracyclines are red aromatic polyketides and occur in variety of forms due to the structural differences in the aglycone and the different sugar residues attached. Anthracyclines are anti-tumor antibiotics that interfere with enzymes involved in DNA replication. These drugs work in all phases of the cell cycle. They are widely used for a variety of cancers. A major consideration when giving these drugs is that they can permanently damage the heart if given in high doses. For this reason, lifetime dose limits are often placed on these drugs. Mitoxantrone is an anti-tumor antibiotic that is similar to doxorubicin in many ways, including the potential for damaging the heart. This drug also acts as a topoisomerase II inhibitor can lead to treatment-

related leukemia. Mitoxantrone is used to treat prostate cancer, breast cancer, lymphoma, and leukemia.

Major drugs in the class are

Daunorubicin : Daunomycin (daunorubicin) was the first anthracycline compound to be characterized structurally and stereochemically. Daunorubicin is used in treating acute lymphoblastic and myeloblastic leukaemias.

Doxorubicin : Doxorubicin is one of the most widely used chemotherapeutic agents and is generally prescribed in combination with other drugs. Doxorubicin has a broad spectrum of activity. It is one of the most effective drugs for solid tumor treatment, e.g., breast cancer, small cell lung cancer and ovarian carcinoma. It has significant activity against bladder, stomach, liver and thyroid tumors, Ewings and osteogenic bone tumors, soft tissue sarcoma, neuroblastoma and Wilms tumor. It is also active against multiple myeloma, several types of leukaemia and cutaneous T-cell lymphoma. It also plays an important role in treatment of Hodgkins and non-Hodgkins lymphomas. The development of resistance in the tumor cells to daunorubicin and doxorubicin, dose dependent cardiotoxicity, and several other side-effects, modification of these drugs to produce analogs with wider activity and lower toxicity have been sought. However, only few anthracycline analogs like epirubicin and idarubicin have been approved for clinical use.

Epirubicin : Epirubicin is an epimer of doxorubicin and differs only in the orientation of the C-4 hydroxyl group on the sugar. This slight change in the structure, epirubicin has lower cardiotoxicity than doxorubicin. Epirubicin is used in the treatment of gastric and breast cancer and is also indicated for the treatment of carcinoid, endometrial, lung, ovarian, esophageal and prostate cancers as well as soft tissue sarcomas.

Idarubicin : Idarubicin is an analog of daunorubicin. It lacks the C-4 methoxy group and this increases its lipophilicity. Idarubicin has improved activity as induction therapy for acute myelogenous leukaemia.

Valrubicin : Valrubicin is N-trifluoroacetyl, 1-4-valerate derivative of doxorubicin. Valrubicin enters cells more rapidly than doxorubicin. It is used specifically in the treatment of early bladder cancer.

Mechanism of action : The selective transport of anthracyclines to the nuclei of neoplastic and proliferating cells has been proposed by Kiyomiya and colleagues. The mechanism by which anthracyclines inhibit cancer is still not completely clear and multiple pathways are thought to be involved. The useful pathways are the ones that are actually involved in toxicity to the neoplasia while not being toxic to the organism. Anthracyclines enter the cells through passive diffusion. It has been demonstrated with doxorubicin that once it enters the cells, it binds the proteasomes in the cytoplasm for which it has high affinity. The drug-proteasome complex is then translocated into the nucleus. Proteasomes are shown to be located predominantly in the nucleus of neoplastic and normal proliferative cells as compared to the

non-proliferative normal cells that show the presence of proteasomes predominantly in the cytoplasm. Thus there will be a relatively higher transport of anthracyclines into the nucleus of the neoplastic and non-differentiated, proliferative normal cells. Once the anthracyclines reach the nucleus they would dissociate from the proteasome and bind DNA due to its higher affinity for DNA. This would bring about the DNA mediated effects of anthracyclines. Moreover, binding of anthracyclines to proteasomes also inhibits the protease activity leading to inhibition of degradation of proteins involved in cell growth and metabolism and thus inducing apoptosis of these cells.

DNA intercalation : Intercalation into DNA leading to inhibition of macromolecular synthesis was the first mechanism described for cytotoxicity of anthracyclines. The rather strong binding of daunorubicin and doxorubicin to DNA has been characterized extensively. However, it has been seen with other anthracyclines nogalamycin family that the antitumor activity correlates with a decrease in affinity for DNA. Considering this DNA intercalation is the only or most essential pathway of anthracycline cytotoxicity. On the other hand, anthracyclines like doxorubicin at low concentrations have been shown to selectively displace nuclear proteins and daunorubicin has been shown to induce aggregation of chromatin. The proposed mechanism involves initial intercalation of the drug into the linker regions where the DNA is free of nuclear proteins, leading to conformational changes in DNA that extend towards the histone octamer and result in the unfolding of chromatin and its subsequent aggregation.

Interaction with DNA binding proteins : Regulation of gene expression by inhibiting, or promoting, the binding of transcription factors is also considered to play a role in anthracycline cytotoxicity with the potential involvement of SP-1 transcription factor as a specific target for these drugs. Involvement of anthracyclines in inhibiting DNA synthesis by affecting the initiation or the elongation phase, and RNA synthesis by inhibiting RNA polymerase activity has also been documented. Another mechanism that has gained ground is the anthracycline activity as Topoisomerase II inhibitor. After DNA intercalation, anthracycline rings that do not intercalate into the DNA seem to play a role in stabilizing the complex between Topoisomerase II and the DNA that it has nicked. The DNA nicks cannot be sealed and this leads to an accumulation of DNA damage that is cytotoxic due to growth arrest in G1 and G2 and programmed cell death. Doxorubicin and Idarubicin have also been showed to inhibit Topoisomerase I and this is proposed to be an ancillary mechanism of cytotoxic activity of anthracyclines.

Anthracyclines, p53 and apoptosis : In some forms of apoptosis p53 played most important role. Like any other genotoxic agent, doxorubicin has been inducing the binding of p53 to DNA. It has been proposed that anthracyclines may exert their cytotoxic effect via p53 mediated apoptosis. There are contradictory reports regarding this link between anthracyclines, p53 and apoptosis. It is observed that there are more DNA breaks in p53 proficient cells than in p53 deficient cells although the levels of Topoisomerase II are same

in the two cell types. It is therefore also proposed that p53 exerts this activity by binding to Topoisomerase II and inhibiting its ligase activity. However, clinical concentrations of these drugs induce apoptosis pathways that do not always require p53 by triggering a cyclic cascade of sphingomyelin hydrolysis and formation of ceramide. It is also observed that anthracyclines can release cytochrome C from mitochondria directly and induce apoptosis. Thus, p53 seems to play some role in the activity of anthracyclines, it is not necessarily the only mechanism.

Free radical generation : One electron addition to the quinone moiety in ring C of anthracyclines leads to formation of semiquinone that regenerates back to quinone by reducing oxygen to reactive oxygen species like superoxide anion and hydrogen peroxide. The semiquinone can oxidize with the bond between ring A and daunosamine which results in deglycosylation. The aglycone thus formed has higher solubility in lipids and can intercalate into biological membranes and form reactive oxygen species which can affect sensitive targets. The one electron redox cycle of doxorubicin has been demonstrated to induce the release of iron from the stores. Doxorubicin forms complex with iron and this complex is capable of producing hydroxyl ions which is a more potent reactive oxygen species. Thus, there seems to be involvement of oxidative damage in the mechanism of anthracycline activity. However, as the production of measurable reactive oxygen species is predominantly observed at supraclinical concentrations, this may not be the direct mechanism of anthracycline activity. Reactive oxygen species though can act as signaling molecules at very low, unmeasurable concentrations and induce apoptosis and this could be one of the mechanism by which anthracyclines exert their cytotoxic effect.

Antiangiogenic mechanism : A recent study shows that the anthracyclines inhibit transcriptional factor HIF-1 from binding to DNA in hypoxic human cells and inhibited tumor growth in human prostate cancer xenografts. Inhibition of HIF-1 transcriptional activity leads to decreased VEGF, SDF1 and SCF expression because of which there is decreased CAC mobilization and this results in decreased tumor vascularization and growth. Thus, anthracyclines can also inhibit cell growth through antiangiogenic pathways.

The side effects of anthracyclines, like any other chemotherapeutic agent, are linked to their cytotoxicity to non-differentiated, proliferating normal cells. These side effects include nausea, vomiting, and alopecia. However, the major toxicities of anthracyclines include cardiotoxicity and myelosuppression and these are the major limitations of these drugs. Doxorubicin can also cause severe local tissue necrosis. Cardiomyopathy and congestive heart failure are the two cardiotoxic side effects of anthracyclines. Epirubicin is less cardiotoxic than doxorubicin but may not totally eliminate the risk of chronic cardiotoxicity. Anthracycline induced cardiotoxicity is irreversible and is thus an especially important consideration in treatment of curative malignancies in pediatric patients. As discussed in the mechanism of action of anthracyclines, the interaction between anthracyclines and iron is found to play a role in anthracycline induced cardiomyopathies by producing potent reactive oxygen species. An iron chelator dexrazoxane has now been approved for use in patients who are

prescribed high doses of doxorubicin to prevent cardiotoxicity. Various other strategies to prevent the cardiotoxicity of anthracyclines are also being employed that include change in administration of the drugs, limiting the overall dosage, encapsulation into liposomes, combination treatment, use of cardioprotectors and synthesis of modified anthracyclines.

Topoisomerase inhibitors : The development of innovative chemotherapeutic treatments for cancer has made great strides within the last few decades. DNA, which is the genetic material, and the enzymes responsible for all the reactions in the body are major targets of interest for medical researchers looking for new cancer treatments. DNA is the blueprint for humans and all other life forms, any damage to the DNA can result in cancer. Enzymes are proteins that facilitate and regulate many processes in the cells, several of which are necessary for the well being and the survival of every individual.

DNA is normally a coiled double helix of two strands and is periodically uncoiled in the process of replication during cell division or in the process of reading the code to make new proteins. Two enzymes that play the biggest role in this uncoiling and recoiling process are topoisomerase I and topoisomerase II. They also play a significant role in fixing DNA damage that occurs as a result of exposure to harmful chemicals or UV rays. There is a distinct difference in way the two enzymes work. Topoisomerase I cuts a single strand of the DNA double helix while topoisomerase II cuts both strands of DNA, using ATP for energy. The rest of the process by which the two enzymes work is very similar. The process entails the relaxation of the coil of the two DNA strands, and then after the cuts are made and replication or repair is complete, the strands are paired back together and reform a coil.

The topoisomerase enzymes have been researched as targets for the generation of new cancer treatments because when they are inhibited in a cell, the result is that the cell dies. Therefore inhibitors of the topoisomerase enzymes have the ability to kill all cells undergoing DNA replication, reading of the DNA for protein production or experiencing repair of DNA damage. Since cancer cells divide much more rapidly than normal cells, the cancer cells will be killed by the topoisomerase inhibitors, though some normal cells with topoisomerase activity will also be killed. The typical way that both topoisomerase I and II inhibitors work is that the inhibitor binds to the topoisomerase molecule. This makes the enzyme nonfunctional by blocking the ability of the topoisomerase to bind the DNA back together after it has been cut. Therefore cuts are made to either one or both strands of the DNA molecule which are never repaired, ultimately leading to death of the cell. Topoisomerase I inhibitors are camptothecin and its derivatives. Topoisomerase II inhibitors include doxorubicin, etoposides and mitoxantrone. Advances have made it possible to treat cancer on a cellular level with more precision and effectiveness than ever before. Inhibiting the topoisomerase enzymes lead to cell death, especially in the cancer cells since they are rapidly dividing. Therefore topoisomerase inhibitors can eradicate certain forms of rapidly growing cancers.

Kinase Inhibitors : Tyrosine kinase inhibitors (TKIs) are a class of chemotherapy medications that inhibit, or block, the enzyme tyrosine kinase. TKIs were created out of modern

genetics- the understanding of DNA, the cell cycle, and molecular signaling pathways- and thus represent a change from general to molecular methods of cancer treatment. This allows for targeted treatment of specific cancers, which lessens the risk of damage to healthy cells and increases treatment success. Receptor Tyrosine Kinases (RTKs) are a family of tyrosine protein kinases. RTKs span the cell membrane with an intracellular (internal) and extracellular (external) portion. The intracellular portion removes a phosphate group, a process called phosphorylation, from the coenzyme messenger ATP. The extracellular portion has sites to which signal sending proteins and hormones can bind. Many of these signaling binders are growth factors. Growth factors are involved in the initialization and regulation of cell cycles. The type of growth factor determines its effects on the cell. There are three primary growth factors that relate to tyrosine kinase. The receptors of these growth factors are members of the RTK family. Epidermal growth factors (EGF) help regulate cell growth and differentiation. Platelet-derived growth factor (PDGF) regulates cell growth and development. Vascular endothelial growth factors (VEGFR) are involved in the creation of blood vessels.

The growth factors, and the kinases, act as though they are attached to an “on/off” switch. The removal of a phosphate group changes the shape and actions of the protein. This essentially “turns on” the cellular action (or actions). When the cellular action(s) is completed, the phosphate group is removed and that protein is “turned off”. This “on/off” process can become disrupted, often thanks to a mutated kinase, and actions can become unregulated. An unregulated RTK bound to EGF, for example, could lead to uncontrolled growth and division in the cell. The rapid cell growth could then lead to cancer. Mutations of RTKs often lead to oncogenes, which are genes that help turn a healthy cell into a cancerous cell. Tyrosine kinase inhibitors treat cancer by correcting this deregulation. How this is done varies depending on the medication, for example Imatinib (brand name: Gleevac) blocks a kinase gene from binding to ATP, preventing the phosphorylation that would benefit the cancerous cell and promote cell division. The medication gefitinib (Iressa) inhibits EGFRs, preventing that signal from being stuck “on” and creating uncontrolled proliferation. Eight TKI medications, including imatinib and gefitinib, have been approved by the Food and Drug Administration (FDA) for use in humans. One TKI, toceranib (Palladia), was recently approved for the treatment of cancer in dogs. The human medications may inhibit one or more tyrosine kinases. Erlotinib (Tarceva), Erlotinib like gefitinib, inhibits EGFR. Lapatinib (Tykerb) is a dual inhibitor of EGFR and a subclass called Human EGFR type 2. EGFR isn’t the only growth factor targeted. Sunitinib (Sutent) is multi-targeted, inhibiting PDGFR and VEGF. Other tyrosine kinase inhibitors are more specialized. Sorafenib (Nexavar) targets a complex pathway that would lead to a kinase signaling cascade. Nilotinib (Tasinga) inhibits the fusion protein bcr-abl and is typically prescribed when a patient has shown resistance to imatinib. Three TKIs are currently showing promise in clinical trials. Bosutinib targets abl and src kinases. Neratinib, like lapatinib, inhibits EGFR and Human EGFR type 2. Vatalanib inhibits both VEGFR and PDGFR. Scientific research is being focused on TKIs because of their uniqueness compared to previous chemotherapy methods. All chemotherapy drugs

seek to stop cells division and growth. They also attempt to kill cancerous cells without destroying the healthy cells. An inherent weakness in cancerous cells is that a failure of cell repair mechanisms is what turned the cells cancerous. The cell is therefore unable to repair damaged or changed DNA effectively. Radiation therapy treatments interfered with the DNA to create a problem the cancer cell cannot repair, leading to cell death. This interference can be done by modifying the building blocks, the bases, by adding to them to prevent bonding. It may be done through mechanical processes that separate the strands of DNA. These methods are rather non-specific, both in terms of the cancers they treat and the cells they act upon. This means that healthy cells may also pay the price of treatment. Destruction of healthy cells is one of the main problems with traditional chemo treatments. TKIs, however, are targeted in that the different types can be cancer-specific, acting upon pathways that have gone awry in the specific cancer. Furthermore, it is often a case that the kinase itself is slightly different from the normal version, meaning that the inhibitor can work specifically on cancerous cells. The only other current treatment that works in a similar way is using monoclonal antibodies, which can be used to target cancerous cells in preference over healthy ones. The specificity of TKIs means there will be fewer side effects and less time in the hospital needed for the patient. The genetic basis for these cancers that can be treated with TKIs can now be screened effectively and early treatment is by far the most effective.

Hormonal Agents : There are two types of hormonal agents used in the treatment of cancer: Corticosteroid hormones and sex hormones. Corticosteroids are used to treat some cancers (leukemia, multiple myeloma, and lymphoma). Steroids are also used to reduce swelling around tumors of the brain and spinal cord. Steroids are used with other chemotherapy drugs in combination chemotherapy. Examples of corticosteroids are Prednisone and Dexamethasone. Sex hormones change how female and male hormones are made and act. They can be used to control the growth of breast, uterine and prostate cancers, which may grow when around hormones. These drugs do not kill cells, as typical chemo drugs do. They cut off the “food supply” to destroy the cancer cells. Examples of sex hormones are Tamoxifen and Leuprolide.

Biological Response Modifiers are drugs that strengthen the bodies’ immune system to fight the growth of cancer. Other agents might stop or slow cancer growth by disrupting processes that are needed to grow or spread. This growing group of anti-cancer medicines is often considered separate from chemotherapy. Examples of biological response modifiers are Herceptin and Avastin, Erbitux and Rituxan.

Miscellaneous Antineoplastics : Several useful types of chemotherapy drugs are unique such as:

- **Ribonucleotide reductase inhibitor :** Hydroxyurea.
- **Adrenocortical steroid inhibitor :** Mitotane
- **Enzymes :** Asparaginase and Pegaspargase.
- **Antimicrotubule agent :** Estramustine

- **Retinoids** : Bexarotene, Isotretinoin, Tretinoin (ATRA)

Beyond the aforementioned types of chemotherapy, many other types of chemo treatments exist, such as targeted therapy, immunotherapy, and hormone therapy.

Other types of cancer drugs : Other drugs and biological treatments are used to treat cancer, but are not usually considered chemotherapy. While chemotherapy drugs take advantage of the fact that cancer cells divide rapidly, these other drugs target different properties that set cancer cells apart from normal cells. They often have less serious side effects than those commonly caused by chemotherapy drugs because they are targeted to work mainly on cancer cells, not normal, healthy cells. Many are used along with chemotherapy.

Targeted therapies : As researchers have learnt more about the inner workings of cancer cells, they have begun to create new drugs that attack cancer cells more specifically than traditional chemotherapy drugs. Most attack cells with mutant versions of certain genes, or cells that express too many copies of a particular gene. These drugs can be used as part of the main treatment, or they may be used after treatment to maintain remission or decrease the chance of recurrence. Examples of targeted therapies include imatinib (Gleevec®), gefitinib (Iressa®), sunitinib (Sutent®) and bortezomib (Velcade®). Targeted therapies are a huge research focus and probably many more will be developed in the future.

Differentiating agents : These drugs act on the cancer cells to make them mature into normal cells. Examples include the retinoids, tretinoin (ATRA or Atralin®) and bexarotene (Targretin®), as well as arsenic trioxide (Arsenox®).

Hormone therapy : Drugs in this category are sex hormones, or hormone-like drugs, that change the action or production of female or male hormones. They are used to slow the growth of breast, prostate, and endometrial (uterine) cancers, which normally grow in response to natural hormones in the body. These cancer treatment hormones do not work in the same ways as standard chemotherapy drugs, but rather by preventing the cancer cell from using the hormone it needs to grow, or by preventing the bodies from making the hormones. Examples include:

- **The anti-estrogens** : fulvestrant (Faslodex®), tamoxifen, and toremifene (Fareston®)
- **Aromatase inhibitors** : anastrozole (Arimidex®), exemestane (Aromasin®), and letrozole (Femara®)
- **Progestins** : megestrol acetate (Megace®)
- Estrogens
- **Anti-androgens** : bicalutamide (Casodex®), flutamide (Eulexin®), and nilutamide (Nilandron®)
- Gonadotropin-releasing hormone (GnRH), also known as luteinizing hormone-releasing hormone (LHRH) agonists or analogs: leuprolide (Lupron®) and goserelin (Zoladex®)

Immunotherapy : Some drugs are given to people with cancer to stimulate their

natural immune systems to recognize and attack cancer cells. These drugs offer a unique method of treatment, and are often considered to be separate from chemotherapy. Compared with other forms of cancer treatment such as surgery, radiation therapy, or chemotherapy, immunotherapy is still fairly new. There are different types of immunotherapy. Active immunotherapies stimulate the body's own immune system to fight the disease. Passive immunotherapies do not rely on the body to attack the disease; instead, they use immune system components (such as antibodies) created outside the body.

Types of immunotherapies and some examples:

- Monoclonal antibody therapy (passive immunotherapies), such as rituximab (Rituxan®) and alemtuzumab (Campath®)
- Non-specific immunotherapies and adjuvants (other substances or cells that boost the immune response), such as BCG, interleukin-2 (IL-2), and interferon-alfa
- Immunomodulating drugs, for instance, thalidomide and lenalidomide (Revlimid®)
- Cancer vaccines (active specific immunotherapies). In 2010, the FDA approved the first vaccine to treat cancer (the Provenge® vaccine for advanced prostate cancer); other vaccines for many different types of cancer are being studied

Process of chemotherapy used:

Sometimes, chemotherapy is used as the only cancer treatment. But more often, chemotherapy will be used along with surgery, radiation therapy, or biological therapy. Chemotherapy can:

- Make a tumor smaller before surgery or radiation therapy. This is called neo-adjuvant chemotherapy.
- Destroy cancer cells that may remain after surgery or radiation therapy. This is called adjuvant chemotherapy.
- Help radiation therapy and biological therapy work properly.
- Destroy cancer cells that have come back (recurrent cancer) or spread to other parts of the body (metastatic cancer).

Mechanism of chemotherapeutic drugs:

When body cells are damaged or die produce new ones to replace dead cells. This is done in an orderly way, in a balanced way. Cancer cells do not have that orderly capacity - their reproduction (division and growth) is out of control - more and more of them are produced and they start to occupy more and more space, until eventually they push out space occupied by useful cells. Chemotherapy drugs interfere with a cancer cell's ability to divide and reproduce. Chemo drugs may be applied into the bloodstream to attack cancer cells throughout the body, or they can be delivered directly to specific cancer sites. Chemotherapy drugs work in various ways such as

- **Impairing mitosis (prevent cell division)** - these are known as cytotoxic drugs.

- Targeting cancer cell's food source, enzymes and hormones they require in order growing.
- Stopping the growth of new blood vessels that supply a tumor.
- Triggering suicide of cancer cells - cell suicide is known medically as apoptosis.

Different stages of Chemotherapy

Neo-adjuvant therapy - if the tumor is large, the surgeon may want to shrink it before surgery. This may involve some pre-operative chemotherapy and/or radiotherapy.

- **Chemoradiation therapy** - the chemotherapy is given in combination with radiotherapy. Patients with localized Hodgkin's lymphoma where the tumor is situated above the diaphragm should be given chemotherapy combined with radiotherapy, European scientists reported after carrying out a clinical trial. Another study reported that the solid tumor cells that survive chemoradiation therapy often end up stronger than they were before.
- **Adjuvant therapy**- chemotherapy is given after surgery. The use of chemotherapy following surgery reduces the risk of death from operable pancreatic cancer by around 30%, a UK study found. Age will determine whether chemotherapy should be used at all for patients with certain cancers. Researchers at The Mayo Clinic, USA, found that the combination of chemotherapies 5FU and oxaliplatin compared to 5FU alone after surgery for colon cancer decreases colon cancer recurrence and promotes longer survival for patients under 70 - but not for those who are older.

A course of chemotherapy may be just a one-day treatment, or can last for a few weeks - it will depend on the type and stage of the cancer. If the patient requires more than one course of treatment, there will be a rest period for his/her body to recover. This could be a one-day treatment followed by a week's rest period, followed by another one-day treatment followed by a three-week rest period, etc. This may be repeated many times. In most countries there will be a multi-disciplinary team who treat the patient's cancer. These may include:

- **A clinical oncologist** - a doctor who specializes in cancer but does not do surgery. He/she is specialized in chemotherapy.
- **A cancer nurse** - probably the first person the patient will meet when coming in for chemotherapy.
- **A hematologist** - this is a doctor who is specialized in the study of blood and bone marrow.
- **A pathologist** - this is a doctor who specializes in the identification of diseases by examining cells and tissues under a microscope.
- **A psychologist** - he/she will help the patient deal with the mental and emotional ordeal of chemotherapy.

Blood tests are needed to assess the health of the patient as well as ensuring that he/

she will be able to cope with possible side-effects. For example, blood tests can detect liver problems, which could mean that chemotherapy is unsuitable for the patient unless the liver recovers. Chemotherapy drugs are metabolized (broken down) in the liver which could be harmed if it is not working properly. Before chemotherapy, it is important to test the patient's blood count because the treatment will reduce the number of red and white blood cells, as well as platelets. If a blood test reveals a low blood count the doctors may decide to delay treatment. Regular blood tests will continue during the treatment period so that the medical team can keep an eye on blood count and the state of the patient's liver. Monitoring the patient's blood can also provide doctors with important data on how well the chemotherapy is working. Two ways of giving chemotherapy depending on the type of cancer, chemotherapy may be administered orally or intravenously (directly into the vein). Oral chemotherapy (swallowing tablets) will be in the form of tablets. If the patient's health allows it he/she will be able to take them at home. However, regular hospital visits will still be needed to check on the patient's health and response to treatment. It is vital that the tablets be taken exactly when specified. If the patient forgets to take one at a specific time he/she should call the medical team immediately. Intravenous chemotherapy (straight into the vein) may be given as an injection straight into a vein, through a drip (intravenous infusion), through a drip or pump. This is called continuous infusion, protracted venous infusion, or ambulant infusion (meaning the patient can walk about while receiving the medication).

There are different ways of getting the medication into the patient. These include:

- **A cannula** - a thin tube is inserted through the skin into the vein - usually it enters the body via the back of the hand or the lower arm.
- **A drip (intravenous infusion)** - in order to dilute the medication it may be injected into a bag. The solution in the bag will pass through a tube into the patient's arm and into a vein (intravenous infusion). A cannula will be used. The solution will enter the vein slowly.
- Chemotherapy through a drip generally is pushed through with a pump. The pump does not hurry the process up, rather it makes sure the solution enters the vein at a constant rate over a specific period - the slower the rate, the longer the whole thing will take.
- **A central line** - this is a long, flexible, plastic line (thin tube) which ends up in a central blood vessel in the chest, close to the heart. The central line usually enters the body through the center of the chest and goes up under the skin into a large vein by the collarbone (clavicle). The only visible part is the length of line that hangs out of the small entry hole in the chest.
- **A peripherally inserted central catheter (PICC) line** - a long, thin, flexible tube that is inserted into a peripheral vein, usually in the upper arm and makes its way into a large vein in the chest near the heart. It is similar to a central line but has a different point of entry.

- **A portacath (implantable port)** - a thin, soft, flexible plastic tube goes into a vein. It has a port (opening) just under the skin of the chest or arm. The port has a thin rubber disc which special needles can pass medicines into, or take blood from.

Side Effects of Chemotherapy : Side effects are problems caused by cancer treatment. Some common side effects from chemotherapy are fatigue, nausea, vomiting, decreased blood cell counts, hair loss, mouth sores, and pain. Chemotherapy is designed to kill fast-growing cancer cells. But it can also affect healthy cells that grow quickly. These include cells that line our mouth and intestines, cells in our bone marrow that make blood cells, and cells that make our hair grow. Chemotherapy causes side effects when it harms these healthy cells.

Although it is an effective treatment for many types of cancer, chemotherapy—like other cancer treatments—often causes side effects. The types and intensity of these side effects vary from person to person and depend on the type and location of cancer, the treatment dose, and the person's overall health. Chemotherapy targets cells that are actively growing and dividing. Although this is a defining characteristic of cancerous cells, it is also a feature of some actively growing normal cells, such as cells in the blood, mouth, intestines, and hair. Side effects occur when the chemotherapy damages these healthy cells that maintain the body's function and appearance.

Doctors and scientists are continually working to identify new drugs, methods of administering (giving) chemotherapy, and combinations of existing treatments that have fewer side effects. As a result, many types of chemotherapy are easier to tolerate than medications used even a few years ago. In addition, doctors have made major strides in recent years in reducing pain, nausea and vomiting, and other physical side effects.

Common side effects of chemotherapy : Different drugs cause different side effects. Although specific side effects may be predictable for certain classes of drugs, each person's experience with chemotherapy is unique. With most types of chemotherapy, the presence and intensity of side effects are not measures of how well the treatment is working. However, some side effects of targeted therapy do, in fact, indicate the medication's effectiveness. Common side effects caused by traditional chemotherapy drugs include:

Fatigue : Most patients receiving chemotherapy will experience some degree of fatigue. This may be a general feeling which exists most of the day, or may only appear after certain activities. Doctors say patients need to make sure they get plenty of rest and not to perform tasks which are overtiring. While light exercise has been shown to help, it is important to remember to keep the activities 'light'. If the tiredness becomes severe it is important to tell the doctor, as this could be caused by a significant drop in red blood cells (anemia).

Pain : Chemotherapy can cause pain for some people, including headaches, muscle pain, stomach pain, and pain from nerve damage, such as burning, numbness, or shooting pains (most often in the fingers and toes). Pain usually diminishes over time, but some people may have symptoms for months or years after chemotherapy has finished due to permanent damage to the nerves. Doctors can manage pain by treating the source of the pain; changing

the perception of pain, usually with pain-relieving medications; or interfering with pain signals sent to the brain through spinal treatments or nerve blocks.

Sores in the mouth and throat : Chemotherapy can damage the cells that line the mouth and throat. The sores (also called mucositis) usually develop five to 14 days after receiving chemotherapy. Although the sores may become infected, they usually heal completely when treatment is finished. Patients receiving chemotherapy who have unhealthy diets and/or poor dental hygiene increase their risk of mouth and throat sores.

Diarrhea : Certain chemotherapy causes loose or watery bowel movements. Preventing diarrhea or treating it early helps a person avoid becoming dehydrated (the condition when the body does not get the amount of fluids it needs) or developing other problems.

Nausea and vomiting : Chemotherapy can cause nausea (an urge to vomit or throw up) and vomiting a risk that depends on the type and dose of chemotherapy. With appropriate medications, nausea and vomiting can be prevented in nearly all patients.

Constipation : Chemotherapy as well as some drugs to treat nausea and vomiting, pain, depression, diarrhea, and high blood pressure may cause constipation (the infrequent or difficult passage of stool). Patients may also increase their risk of constipation by not drinking enough fluids, not eating balanced meals, or not getting enough exercise.

Neutropenia (low white blood cells) - Susceptibility to infections: When receiving chemotherapy the immune system will be weakened because the white blood cell count will go down. White blood cells form part of the immune system - they fight infection. Consequently, patients become more susceptible to infections. Some patients will be prescribed antibiotics which may reduce their risk of developing infections. Patients receiving chemotherapy who develop an infection need immediate treatment. This may mean being hospitalized and receiving antibiotics via an intravenous drip.

Thrombocytopenia (low blood platelet count) - Blood clotting problems: Chemotherapy may lower the patient's blood platelet count. A platelet is a type of blood cell that helps the blood to clot (coagulate). Coagulation is essential, otherwise bleeding does not stop. Lower blood platelet counts linked to chemotherapy is a risk, but less so than lower red or white blood cell counts. If we are affected we will bruise more easily, we will be more likely to have nosebleeds and bleeding gums, and if you cut yourself it may be harder to stop the bleeding.

Anemia (low red blood-cell count) : As well lowering white blood cell count, chemotherapy will also lower red blood cell count. Tissues and organs inside body get their oxygen from the red blood cells. People with anemia feel very tired. A patient on chemotherapy who has anemia will feel extra tired - much more tired than straightforward fatigue caused by the treatment. Dyspnea (shortness of breath) is also another symptom of anemia, as are palpitations (when the heart beat is irregular). Anemia linked to chemotherapy requires immediate treatment. A blood transfusion will bring the red blood cell count back up immediately. Erythropoietin (EPO) is a drug that makes the body produces more red

blood cells.

Nervous system effects : Some drugs cause nerve damage, resulting in one or more of the following nerve- or muscle-related symptoms: Tingling, Burning, Weakness or numbness in the hands and/or feet, Weak, sore, tired, or achy muscles, Loss of balance, Shaking or trembling, Stiff neck, Headache, Visual problems, Walking problems, Difficulty hearing and Clumsiness.

These symptoms usually improve when the chemotherapy dose is lowered or treatment is stopped; however, in some cases, the damage is permanent.

Changes in thinking and memory : Some patients experience difficulty thinking clearly and concentrating after chemotherapy. Cancer survivors often refer to this side effect as “chemo brain,” while doctors may refer to it as cognitive changes or cognitive dysfunction.

Sexual and Reproductive System : Chemotherapy drugs can have an effect on our hormones. In women, hormonal changes can bring on hot flashes, irregular periods, or sudden onset of menopause. They may become temporarily or permanently infertile. Women on chemotherapy may experience dryness of vaginal tissues that can make intercourse uncomfortable or painful. The chance of developing vaginal infections is increased. Chemotherapy drugs given during pregnancy can cause birth defects. In men, some chemo drugs can harm sperm or lower sperm count, and temporary or permanent infertility is possible. In addition, chemotherapy is capable of harming a fetus (unborn baby) during pregnancy, particularly if given during the first trimester of pregnancy when the fetus’ organs are still developing. Women should take precautions to avoid pregnancy during treatment and tell their doctor if they become pregnant.

Appetite loss : People receiving chemotherapy may eat less than usual, not feel hungry at all, or feel full after eating only a small amount. Ongoing appetite loss can lead to weight loss, malnutrition, and loss of muscle mass and strength, which can hinder the body’s ability to recover from chemotherapy. Learn more about managing appetite loss.

Kidneys and Bladder (Excretory System) : The kidneys work to excrete the powerful chemotherapy drugs as they move through our body. In the process, some kidney and bladder cells can become irritated or damaged. Symptoms of kidney damage include decreased urination, swelling of the hands and feet (edema), and headache. Symptoms of bladder irritation include a feeling of burning when urinating and increased urinary frequency.

Alopecia (Hair loss) : Some chemotherapy medications cause hair loss while others don’t. If hair does start to fall out this will usually happen a few weeks after treatment starts. On some occasions the hair will just become thinner and more brittle (without falling out). Hair loss can occur in any part of the body. Although hair loss has no physical health consequences, it may cause distress and embarrassment for some people. The psychological impact tends to be greater among women than men

Long-term side effects : Most side effects of chemotherapy disappear at the end of

treatment. However, some side effects may continue, come back, or develop later. For instance, certain types of chemotherapy are associated with permanent organ damage to the heart, lung, liver, kidneys, or reproductive system. In addition, some people find that cognitive functions (such as thinking, concentrating, and memory) remain a challenge for months or years after treatment. Nervous system changes can also develop after treatment, and children who have received chemotherapy may experience late effects (side effects that occur months or years after cancer treatment). Cancer survivors also have a higher risk of developing second cancers later in life.

Follow-up care is essential for all cancer survivors and may include regular physical examinations and/or medical tests to monitor recovery in the months and years after cancer treatment. ASCO offers cancer treatment summary forms to help keep track of the cancer treatment we received and develop a survivorship care plan once treatment is completed.

GENE THERAPY

Genes are composed of DNA that carries information needed to make proteins – the building blocks of our bodies. Variations in the DNA sequence or code of a gene are called mutations, which often are harmless but sometimes can lead to serious disease. Gene therapy treats disease by “repairing” dysfunctional genes or by providing copies of missing genes. Gene therapy is a novel approach to treat, cure, or ultimately prevent disease by changing the expression of a person’s genes. Gene therapy is in its infancy, and current therapies are primarily experimental, with most human clinical trials still in the research stages. Gene therapy is the use of nucleic acid polymers as a drug to treat disease by therapeutic delivery into a patient’s cells, where they are either expressed as proteins, interfere with the expression of proteins, or possibly even correct genetic mutations. The most common form of gene therapy involves using DNA that encodes a functional, therapeutic gene to replace a mutated gene. In gene therapy, the nucleic acid molecule is packaged within a “vector”, which is used to get the molecule inside cells within the body.

Gene therapy was first conceptualized in 1972, with the authors urging caution before commencing gene therapy studies in humans. The first FDA-approved gene therapy experiment in the United States occurred in 1990, when Ashanti DeSilva was treated for ADA-SCID. In January, 2014, about 2,000 clinical trials had been conducted or had been approved using a number of techniques for gene therapy. Although early clinical failures led many to dismiss gene therapy as over-hyped, clinical successes since 2006 have bolstered new optimism in the promise of gene therapy. These include successful treatment of patients with the retinal disease Leber’s congenital amaurosis, X-linked SCID, ADA-SCID, adrenoleukodystrophy, chronic lymphocytic leukemia (CLL), acute lymphocytic leukemia (ALL), multiple myeloma, haemophilia and Parkinson’s disease. These clinical successes have led to a renewed interest in gene therapy, with several articles in scientific and popular publications calling for continued investment in the field and between 2013 and April 2014, US companies invested over \$600 million in gene therapy. Two main approaches have been considered for gene therapy - adding a gene to replace a gene that wasn’t working properly, or disrupting genes that were not working properly. Scientists focused on diseases caused by single-gene defects, such as cystic fibrosis, hemophilia, muscular dystrophy, thalassemia, and sickle cell anemia. As of 2014, gene therapy was still generally an experimental technique, although in 2012 Glybera became the first gene therapy treatment to be approved for clinical use in either Europe or the United States after its endorsement by the European Commission, as a treatment for a disease caused by a defect in a single gene, lipoprotein lipase. To reverse disease caused by genetic damage, researchers isolate normal DNA and package it into a vehicle known as a vector, which acts as a molecular delivery truck. Vectors composed of viral DNA sequences have been used successfully in human gene therapy trials. Doctors infect a target cell usually from a tissue affected by the illness, such as liver or lung cells with the vector. The vector unloads its

DNA cargo, which then begins producing the proper proteins and restores the cell to normal. Problems can arise if the DNA is inserted into the wrong place in the genome. For example, in rare instances the DNA may be inserted into a regulatory gene, improperly turning it on or off, leading to cancer.

Researchers continue to optimize viral vectors as well as develop non-viral vectors that may have fewer unexpected side effects. Non viral gene delivery involves completing DNA with an agent that allows it to enter a cell nonspecifically. DNA delivered in this manner is usually expressed for only a limited time because it rarely integrates into the host cell genome. Initial efforts in gene therapy focused on delivering a normal copy of a missing or defective gene, but current programs are applying gene delivery technology across a broader spectrum of conditions. Researchers are now utilizing gene therapy to:

- Deliver genes that catalyze the destruction of cancer cells or cause cancer cells to revert back to normal tissue.
- Deliver viral or bacterial genes as a form of vaccination.
- Deliver genes that promote the growth of new tissue or stimulate regeneration of damaged tissue

Several methods such as surgery, radiation, and chemotherapy have been used to treat cancers. The cancer patients who are not helped by these therapies may be treated by gene therapy. Gene therapy is the insertion of a functional gene into the cells of a patient to correct an inborn error of metabolism, to alter or repair an acquired genetic abnormality, and to provide a new function to a cell. In healthy adults, the immune system may recognize and kill the cancer cells; unfortunately, cancer cells can sometimes evade the immune system resulting in expansion and spread of these cancer cells leading to serious life threatening disease. Cancer cells often different from their normal neighbors by a host of specific phenotypic changes, such as rapid division rate, invasion of new cellular territories, high metabolic rate, and altered shape. Some of those mutations may be transmitted from the parents through the germ line. Other arise de novo in the somatic cell lineage of a particular cell.

2. Types of gene therapy:

Two basic types of gene therapy have been applied to human's namely germinal and somatic therapy.

2.1. Germinal gene therapy : In germ line gene therapy, germ cells (sperm or eggs) are modified by the introduction of functional genes, which are integrated into their genomes. Germ cells will combine to form a zygote which will divide to produce all the other cells in an organism and therefore if a germ cell is genetically modified then all the cells in the organism will contain the modified gene. Germ line cells results in permanent changes that are passed down to subsequent generations. If done early in embryologic development, such as during pre implantation diagnosis and in vitro fertilization, the gene transfer could also occur in all cells of the developing embryo. The appeal of germ line gene therapy is its potential for

offering a permanent therapeutic effect for all who inherit the target gene. Successful germ line therapies introduce the possibility of eliminating some diseases from a particular family, and ultimately from the population, forever. However, this also raises controversy. Some people view this type of therapy as unnatural, and liken it to “playing God.” Others have concerns about the technical aspects. They worry that the genetic change propagated by germ line gene therapy may actually be deleterious and harmful, with the potential for unforeseen negative effects on future generations. Some jurisdictions, including Australia, Canada, Germany, Israel, Switzerland, and the Netherlands prohibit this for application in human beings, at least for the present, for technical and ethical reasons, including insufficient knowledge about possible risks to future generations and higher risk than somatic gene therapy (e.g. using non-integrative vectors).

2.2. Somatic gene therapy : Somatic cells are non reproductive. Somatic cell therapy is viewed as a more conservative, safer approach because it affects only the targeted cells in the patient, and is not passed on to future generations. In other words, the therapeutic effect ends with the individual who receives the therapy. However, this type of therapy presents unique problems of its own. Often the effects of somatic cell therapy are short-lived. Because the cells of most tissues ultimately die and are replaced by new cells, repeated treatments over the course of the individual’s life span are required to maintain the therapeutic effect. Transporting the gene to the target cells or tissue is also problematic. Regardless of these difficulties, however, somatic cell gene therapy is appropriate and acceptable for many disorders, including cystic fibrosis, muscular dystrophy, cancer, and certain infectious diseases. Clinicians can even perform this therapy in utero, potentially correcting or treating a life-threatening disorder that may significantly impair a baby’s health or development if not treated before birth.

The results of any somatic gene therapy are restricted to the actual patient and are not passed on to his or her children. All gene therapy to date on humans has been directed at somatic cells, whereas germline engineering in humans remains controversial and prohibited in for instance the European Union. Somatic gene therapy can be broadly split into two categories:

- Ex vivo, which means exterior (where cells are modified outside the body and then transplanted back in again). In some gene therapy clinical trials, cells from the patient’s blood or bone marrow are removed and grown in the laboratory. The cells are exposed to the virus that is carrying the desired gene. The virus enters the cells and inserts the desired gene into the cells’ DNA. The cells grow in the laboratory and are then returned to the patient by injection into a vein. This type of gene therapy is called ex vivo because the cells are treated outside the body.
- In vivo, which means interior (where genes are changed in cells still in the body). This form of gene therapy is called in vivo, because the gene is transferred to cells inside the patient’s body.

One of the most promising approaches to emerge from the improved understanding

of cancer at the molecular level is the possibility of using gene therapy to selectively target and destroy tumor cells, for example, the loss of tumor suppressor genes (e.g. p53 gene) and the over expression of oncogenes (e.g. K-RAS) that have been identified in a number of malignancies. It may be possible to correct an abnormality in a tumor suppressor gene such as p53 by inserting a copy of the wild-type gene; in fact, insertion of the wild-type p53 gene into p53-deficient tumor cells has been shown to result in the death of tumor cells. This has significant implications, since p53 alterations are the most common genetic abnormalities in human cancers. The over expression of an oncogene such as K-RAS can be blocked at the genetic level by integration of an antisense gene whose transcript binds specifically to the oncogene RNA, disabling its capacity to produce protein. Experiments in vitro and in vivo have demonstrated that when an antisense K-RAS vector is integrated into lung cancer cells that over express K-RAS their tumor genicity is decreased.

Despite the promise of such approaches, a number of difficulties remain to be overcome, the most important of which is the need for more efficient systems of gene delivery. No gene transfer system is 100% efficient, unless germ-line therapy is contemplated. During the past two decades, there have been major advances in our understanding of how cancer develops, proving that cancer has a genetic basis. A series of genetic abnormalities that accumulate in one cell may result in a pattern of abnormal clonal proliferation. Our growing understanding of the genetic basis of cancer offers new opportunities for the molecular prevention and treatment of cancer. There has been a substantial growth in gene therapy, especially in the field of oncology since the first experiment in human gene therapy began in 1990, with the aim of treating adenosine deaminase deficiency. By the end of 1993, there were 45 approved trials by US Recombinant DNA Advisory Committee, 30 of which are for the treatment of cancer. This is, in part, because tumor cells can be manipulated ex vivo, while the affected tissues from individuals with other genetic diseases often cannot.

More than 100 clinical applications of gene transfer into human patients for both therapeutic and cell-marking purposes have now been approved in the USA and a number of other countries. Trials for cancer gene therapy that have been approved in the USA have involved malignancies that are considered incurable. This clinical situation, which is unlike many genetic diseases for which life expectancy is measured in years rather than weeks or months, has been considered more appropriate ethically for untested technologies. For these reasons, applications of gene therapy to cancer will continue to be the fastest growing area of human gene therapy.

3. Vectors in gene therapy:

Gene therapy utilizes the delivery of DNA into cells, which can be accomplished by a number of methods. The two major classes of methods are those that use recombinant viruses (sometimes called biological nano particles or viral vectors) and those that use naked DNA or DNA complexes (non-viral methods).

3.1. Viruses : All viruses bind to their hosts and introduce their genetic material into

the host cell as part of their replication cycle. Therefore this has been recognized as a plausible strategy for gene therapy, by removing the viral DNA and using the virus as a vehicle to deliver the therapeutic DNA. A number of viruses have been used for human gene therapy, including retrovirus, adenovirus, lenti virus, herpes simplex virus, vaccinia, pox virus and adeno-associated virus.

3.2. Non-viral methods : Non-viral methods can present certain advantages over viral methods, such as large scale production and low host immunogenicity. Previously, low levels of transfection and expression of the gene held non-viral methods at a disadvantage; however, recent advances in vector technology have yielded molecules and techniques that approach the transfection efficiencies of viruses. There are several methods for non-viral gene therapy, including the injection of naked DNA, electroporation, the gene gun, sonoporation, magnetofection, and the use of oligonucleotides, lipoplexes, dendrimers, and inorganic nanoparticles.

4. Procedure of Gene therapy:

One of the most exciting treatments to emerge from the concept of gene therapy is that of gene transfer or insertion. This is a radically new treatment paradigm involving the introduction of a foreign gene into the cancer cell or surrounding tissue. Genes with a number of different functions have been proposed for this type of therapy, including suicide genes (genes that cause cellular death when expressed), anti angiogenesis genes and cellular stasis genes. A number of different viral vectors have been used in clinical trials to deliver these genes, but most commonly have used a replication incompetent adenovirus. Nonviral methods, including naked DNA transfer and oligodendromer DNA coatings, as well as electroporation are also viable modes of gene delivery. The type of delivery vehicle chosen depends on the desired specificity of the gene transfer therapy, as well as the length of time the gene must be expressed in order to be effective. For instance, a replication incompetent adenoviral vector containing the herpes simplex virus thymidine kinase (HSVtk) gene needs only transient expression to accomplish cell death and is generally delivered via an adenoviral vector. However, anti angiogenesis genes, such as sFLT-1 and statin-AE, need continuous expression for therapeutic effect and have been delivered using plasmids that contain transposons to insert the gene into the cellular DNA. Initial attempts to implement gene transfer therapy have highlighted its promise, as well as some delivery difficulties.

Delivery of the therapeutic gene to the target cells has to be effective enough to elicit a response and has been difficult to achieve with many of the current technologies. In addition, extra precautions must be taken to ensure the therapeutic gene does not integrate into unwanted cell types, such as reproductive tissues. Earlier gene transfer trials suffered from gene silencing so that even if the gene was effectively introduced into the cell, it was not expressed or was expressed only for a limited length of time. Despite these hurdles, solid tumors such as prostate, lung and pancreatic tumors have been treated successfully in animal models using a variety of genes and transfer methods. Special precautions must be taken if DNA is inserted

into the cell chromosome. The insertion site must be in an area of the genome that does not promote cancer. Retro transposon, such as sleeping beauty (an artificially constructed retro transposon that is used to insert genes into vertebrate chromosomes) often insert into actively transcribed genes causing potential problems for cellular function. Preclinical models using gene insertion techniques, such as murine models for glioma, showed significantly greater survival when they administered antiangiogenic genes via a retrotransposon system injected intracranially.

Gene therapy is designed to introduce genetic material into cells to compensate for abnormal genes or to make a beneficial protein. If a mutated gene causes a necessary protein to be faulty or missing, gene therapy may be able to introduce a normal copy of the gene to restore the function of the protein. A gene that is inserted directly into a cell usually does not function. Instead, a carrier called a vector is genetically engineered to deliver the gene. Certain viruses are often used as vectors because they can deliver the new gene by infecting the cell. The viruses are modified so they can't cause disease when used in people. Some types of virus, such as retroviruses, integrate their genetic material (including the new gene) into a chromosome in the human cell. Other viruses, such as adenoviruses, introduce their DNA into the nucleus of the cell, but the DNA is not integrated into a chromosome. The vector can be injected or given intravenously (by IV) directly into a specific tissue in the body, where it is taken up by individual cells. Alternately, a sample of the patient's cells can be removed and exposed to the vector in a laboratory setting. The cells containing the vector are then returned to the patient. If the treatment is successful, the new gene delivered by the vector will make a functioning protein. Researchers must overcome many technical challenges before gene therapy will be a practical approach to treating disease. For example, scientists must find better ways to deliver genes and target them to particular cells. They must also ensure that new genes are precisely controlled by the body.

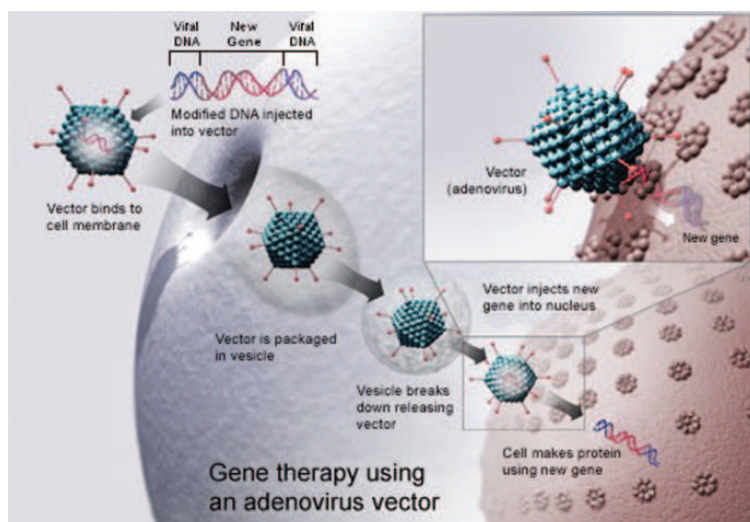


Fig: A new gene is injected into an adenovirus vector, which is used to introduce the modified DNA into a human cell. If the treatment is successful, the new gene will make a functional protein.

4.1. Current Clinical Trials:

TNFERade is one such treatment option that is currently in late stage II trials. This agent is a replication incompetent adenoviral vector that delivers the tumor necrosis factor- α (TNF- α) gene under the transcriptional control of a radiation inducible promoter. TNF- α is a cytokine with potent anticancer properties and high systemic toxicity, and TNF- α gene therapy provides a way to target this molecule to only the cancer cells through the use of intra tumoral injections and a promoter that is activated by radiation therapy. Once TNFERade is injected, the patient then receives radiation therapy to the tumor to activate the gene. The gene then produces the TNF- α molecule which in combination with the radiation therapy promotes cell death in the affected cancer cells and surrounding cells. A phase I study of patients with soft tissue sarcoma using TNFERade demonstrated an 85% response rate including 2 complete responses. In another large phase I study of patients with histologically confirmed advanced cancer, 43% of the patients demonstrated an objective response with 5 of 30 exhibiting complete response to the treatment. Larger studies are being conducted using TNFERade for treatment in pancreatic, esophageal, rectal cancer and melanoma. Another exciting gene therapy treatment agent is Rexin-G, the first injectable gene therapy agent to achieve orphan drug status from the Food and Drug Administration for treatment of pancreatic cancer. This gene therapy agent contains a gene designed to interfere with the cyclin G1 gene and is delivered via a retroviral vector. The gene integrates into the cancer cell's DNA to disrupt the cyclin G1 gene and causes cell death or growth arrest. In a phase I trial, 3 of 3 patients experienced tumor growth arrest with 2 patients experiencing stable disease. These results have led to larger phase I and II trials. Rexin-G is also being evaluated for colon cancer that has metastasized to the liver. A gene transfer technology that shows great promise is the replication incompetent adenovirus delivering the HSVtk gene to a tumor followed by ganciclovir treatment. Ganciclovir is not toxic unless metabolized by the HSVtk gene, and therefore only the cancer cells that are treated with the gene and the surrounding cells will be affected by treatment. In a large phase I study involving glioblastoma patients, the HSVtk-engineered viral treatment increased median survival from 39 weeks to 70.6 weeks and was the first glioblastoma gene therapy trial to show any measurable improvement in several agents that use a replication incompetent adenoviral vector to deliver the p53 gene to cancer cells are also currently in phase II and III trials. The p53 gene is an important cell cycle regulator that has been extensively studied and is mutated in 50% to 70% of human tumors. Mutations in this gene are often linked to aggressiveness. It has been shown that restoration of a functional p53 gene in cancer cells results in tumor cell stasis and often apoptosis. Using this information, INGN 201, an adenoviral vector containing p53 for gene transfer, is in current phase III testing for squamous cell carcinoma of the head and neck, and has completed phase I studies on prostate, ovarian, glioma and bladder cancer.

4.2. Future Directions : Gene transfer, while a radical new type of treatment, is also the only gene therapy product to obtain regulatory approval in any global market, as demonstrated by China's 2003 approval of Gendicine for clinical use. Gendicine is a modified

adenovirus that delivers the p53 gene to cancer cells and is approved for the treatment of head and neck squamous cell carcinoma. Since approval, thousands of patients have been treated in China; some with repeated injections. As yet, large-scale efficacy trial results have not been published; the results of which are eagerly awaited. Gene transfer technology allows an incredible diversity of treatment possibilities. This diversity can be used to complement traditional therapies, as well as provide radically new frontiers for treatment. Gene transfer therapy can rely on the current information known about the genetics of cancer formation, bringing a more sophisticated and personalized approach to therapy. Current gene transfer trials have demonstrated statistically significant survival improvements for cancers such as glioblastoma and pancreatic cancer. These studies have provided very encouraging signs that current research is on the right path. New delivery methods and more sophisticated gene expression cassettes will create better therapeutic alternatives to make the goal of cancer treatment and eradication achievable.

5. Gene therapy strategies to correct or eliminate cancer cells:

Cancer causes cells to grow aberrantly. The growth of cancer cells leads to damage of normal tissues, causing loss of function and often pain. Many types of tumors shed cells that migrate to other distant sites in the body, establish a base there, and grow continuously. These secondary cancer sites, called metastases, cause local destruction and loss of normal tissue function. Multiple cumulative mutations are needed to cause cancer. While similar mutations are found in many cancers, no single mutation is found in all cancers. A number of gene therapy strategies are being evaluated in humans with cancer and these include manipulating cells to gain or lose function. For example, half of all cancers have a mutated p53 protein which interferes with the ability of tumor cells to self-destruct by a process called apoptosis. To this end, investigators are currently testing in clinical trials the ability to genetically introduce a normal p53 gene into these cancer cells. Introduction of a normal p53 gene renders the tumor cells more sensitive to standard chemotherapy and radiation treatments compared to tumor cells expressing the abnormal protein. Furthermore, other tumor suppressor genes are being placed in gene cassettes for expression in tumor cells which can similarly render them more sensitive to apoptosis, or the process of programmed cell death. Alternatively, other investigators are utilizing gene therapy approaches to induce expression of immune stimulating proteins called cytokines which in turn may increase the ability of the patient's own immune system to recognize and kill these cancer cells. Another therapeutic approach, termed gene silencing, is designed to inhibit the expression of specific genes which are activated or over expressed in cancer cells and can drive tumor growth, blood vessel formation, seeding of tumor cells to other tissues, and allow for resistance to chemotherapy. Several such proteins, termed oncogenes, increase cell division and are often expressed continuously at high concentrations in cancer cells. Alternatively, as a tumor grows, it requires new blood vessel formation to survive by a process known as angiogenesis, which is mediated by a different set of proteins.

Furthermore, tumor cells have the capacity to travel through the blood and seed other

tissues where they can grow in a process termed transition, once again mediated by a different set of genes. Finally, scientists have identified genes in tumor cells which allow for these tumor cells to escape killing by chemotherapy. Therefore, an alternative gene therapy approach for cancer is to target one or more of these genes in order to suppress or silence their expression resulting in an inability of these tumor cells to maintain cell growth, inhibit metastases, impair blood vessel formation or reverse drug resistance.

Alternatively, gene therapy approaches may be designed to directly kill tumor cells using tumor-killing viruses, or through the introduction of genes termed suicide genes into the tumor cells. Scientists have generated viruses, termed oncolytic viruses, which grow selectively in tumor cells as compared to normal cells. Tumor cells, but not normal cells, infected with these viruses are then selectively killed by the virus. Oncolytic viruses spread deep into tumors to deliver a genetic payload that destroys cancerous cells. Several viruses with oncolytic properties are naturally occurring animal viruses (Newcastle Disease Virus) or are based on an animal virus such as vaccinia virus (cow pox virus or the small pox vaccine). A few human viruses such as coxsackie virus A21 are similarly being tested for these properties. Human viruses such as measles virus, vesicular stomatitis virus, reovirus, adenovirus, and herpes simplex virus (HSV) are genetically modified to grow in tumor cells, but very poorly in normal cells. Currently, multiple clinical trials are recruiting patients to test oncolytic viruses for the treatment of various types of cancers.

Suicide genes encode enzymes that are produced in tumor cells and can convert a nontoxic prodrug into a toxic drug. Examples of suicide enzymes and their prodrugs include HSV thymidine kinase (ganciclovir), *Escherichia coli* purine nucleoside phosphorylase (fludarabine phosphate), cytosine deaminase (5-fluorocytosine), cytochrome p450 (cyclophosphamide), cytochrome p450 reductase (tirapazamine), carboxy peptidase (CMDA), and a fusion protein with cytosine deaminase linked to mutant thymidine kinase. For example, the efficacy of an oncolytic adenovirus which expresses the fused cytosine deaminase linked to mutant thymidine kinase is currently being investigated in a phase II/III trial for the treatment of prostate cancer. Significantly, prior pilot studies indicated that the treatment of the prostate cancer cells with the suicide genes introduced by the oncolytic virus increased cancer cell sensitivity to radiation and chemotherapy.

6. Cellular immune therapy of cancer:

All the above approaches have the limitation that they require delivery of a “corrective” gene to every cancer cell, a demanding task. An alternative is to harness the immune system which may have an ability to actively seek out cancer cells. In healthy adults, the immune system recognizes and kills precancerous cells as well early cancer cells, but as the cancer progresses the immune system can become overwhelmed. In fact, many cancers have an ongoing ability to further inhibit the ability of a patient’s immune system to target and eradicate the tumor cells. To this end, investigators are developing and testing several cell therapy strategies to correct impairment of the patient’s immune system and as a consequence, to improve the

immune system's ability to eliminate cancer.

Cell therapy for cancer refers to one or more of 3 different approaches such as

- Therapy with cells that give rise to a new immune system which may be better able to recognize and kill tumor cells through the infusion of hematopoietic stem cells derived from umbilical cord blood, peripheral blood or bone marrow cells.
- Therapy with immune cells such as dendritic cells which are designed to activate the patient's own resident immune cells (e.g. T cells) to kill tumor cells.
- Direct infusion of immune cells such as T cells and NK cells which are prepared to find, recognize, and kill cancer cells directly.

In all three cases, therapeutic cells are harvested and prepared in the laboratory prior to infusion into the patient. Immune cells including dendritic cells, T cells and NK cells, can be selected for desired properties and grown to high numbers in the laboratory prior to infusion. Challenges with these cellular therapies include the ability of investigators to generate sufficient numbers of cells for therapy without damaging the ability of these cells to participate in clearance of the tumor. For example, it can be a challenge at times to identify and select immune cells that can efficiently find and then kill the tumor cells in the patient while maintaining these qualities until the cancer cells are fully eliminated. Clinical trials of cell therapy for many different cancers are currently ongoing with promising results.

More recently, scientists have developed novel cancer therapies by combining both gene and cell therapies. Specifically, investigators have developed genes which encode for artificial receptors, which, when expressed by immune cells, allow these cells to specifically recognize cancer cells thereby increasing the ability of these gene modified immune cells to kill cancer cells in the patient. One example of this approach, which is currently being studied at multiple centers, is the gene transfer of a class of novel artificial receptors called "chimeric antigen receptors" or CARs for short, into a patient's own immune cells, typically T cells, in the laboratory. The resulting genetically modified T cells which express the CAR gene are now able to recognize and kill tumor cells. Significantly, scientists have developed a large number of CARs which recognize different molecules on different types of cancer cells. For this reason, investigators believe that this approach may hold promise in the future for patients many different types of cancer. To this end, multiple pilot clinical trials for multiple cancers using T cells genetically modified to express tumor specific CARs are in currently enrolling patients and these too show promising results.

Gene therapy for the treatment of cancer has a wide variety of potential uses. There are several potential strategies for gene therapy in the treatment of cancer. Strategies of gene therapy for cancer are

- Enhancing the immunogenicity of the tumor, for example by introducing genes that encode foreign antigens.
- Enhancing immune cells to increase anti-tumor activity, for example by introducing

genes that encode cytokines.

- Inserting a “sensitivity” or suicide’ gene into the tumor, for example by introducing the gene that encodes HSVtk.
- Blocking the expression of oncogenes, for example by introducing the gene that encodes antisense K-RAS message.
- Inserting a wild-type tumor suppressor gene, for example P53 or the gene involved in Wilm’ tumor.
- Protecting stem cells from the toxic effects of chemotherapy, for example by introducing the gene that confers MDR-1.
- Blocking the mechanisms by which tumors evade immunological destruction, for example by introducing the gene that encodes antisense IGF-1 message.
- Killing tumor cells by inserting toxin genes under the control of a tumor-specific promoter, for example the gene that encodes diphtheria A chain.

6. Approaches to ex vivo gene transfer includes

6.1. Genetically engineered tumor cells : Various groups are investigating the production of autologous cellular vaccines for the treatment and prevention of cancer. This is most commonly attempted by surgically removing tumor cells from the patient, growing them in tissue culture and inserting immune stimulatory genes in vitro. These cells are then re injected into the patient in an effort to induce a significant systemic immune response that will both destroy tumor cells and protect the patient against a recurrence of the tumor. Treating cells that produce cytokines has been shown to result in systemic immunity in mice. Alteration of syngeneic tumors with the genes that encode IL-1b, IL-2, IL-4, IL-6, TNF α , GM-CSF or r-interferon results in immunological destruction of the tumor cells in vivo. In human gene therapy trials, patients are injected with either autologous or allogeneic genetically modified tumor cells. These trials involved the insertion of retroviral vectors carrying the gene that encodes IL-2, TNF α or GM-CSF into melanoma, colorectal renal cell carcinoma, neuroblastoma or breast cancer cells in vitro. One modification of this technique is the insertion of the gene for either IL-2 or IL-4 into autologous fibroblasts, which are then mixed with irradiated tumor cells from the patient and re injected. This approach has the advantage that growing fibroblasts in vitro is much easier than culturing tumor cells from a large number of individuals. Besides modifying tumor cells to produce immune activating cytokines, another strategy is to block the production of insulin-like growth factor-1 (IGF-1). Many tumors such as breast cancer produce high levels of IGF-1. Insertion of an antisense gene that stops production of IGF-1 in the tumor allows immunological rejection of the genetically altered tumor after re implantation. Destruction of the tumor is mediated by cytotoxic T lymphocytes. The precise mechanism by which IGF-1 mediates tumor protection in vivo remains unclear.

6.2. Genetically engineered T lymphocytes : T lymphocytes have the capacity to hone

in on tumor tissue. This property has been used to deliver cytokines directly to tumor masses for human gene therapy. The secretion of cytokines locally at the tumor site by the effectors T lymphocytes will enhance their anti-tumor activity and avoid the side-effects that result from the systemic administration of cytokines. For the trial of TNF-modified tumor infiltrating T lymphocytes, T lymphocytes are difficult to transduce with retroviral vectors and tend to down regulate expression of the cytokine gene carried by the vector. These two problems of poor gene transfer efficiency and poor cytokine expression have so far limited the application of this approach, and have shifted the emphasis from modification of T lymphocytes toward the genetic alteration of tumor cells, which are much easier to grow in culture and more readily engineered.

Insertion of a sensitivity gene: Gene therapy uses the genes to activate a relatively nontoxic pro-drug to form a highly toxic agent. The most widely studied system uses the thymidine kinase gene of the Herpes simplex virus (HSVtk). The HSVtk gene confers sensitivity to the anti-herpes drug, ganciclovir (GCV), by phosphorylating GCV to a monophosphate form (GCV-MP). Phosphorylation to the triphosphate form (GCV-TP) by cellular kinases results in inhibition of DNA polymerase, and leads to cell death. In this procedure, GCV kills tumor cells which express KSVtk, and the adjacent cells that lack the gene are also destroyed. This is termed the bystander effect phenomenon. To use the bystander effect to kill human cancer in vivo, the irradiated ovarian tumor cells that contain the HSVtk gene will be injected into the peritoneal cavity of patients, who will be given GCV. These HSVtk-expressing cells will destroy bystander tumor cells in vivo.

6.3. Protection of hematopoietic stem cells : Protection of hematopoietic stem cells (HSCs) from the toxic effects of chemotherapy by using the gene that confers multiple drug resistance type 1(MDR-1) is another possible strategy for human cancer therapy. The MDR-1 gene will be isolated from tumor cells, where it functions to pump chemotherapy drugs (including daunorubicin, doxorubicin, vincristine, vinblastine, VP-16, VM-26, taxol and actinomycin-D) from within the cell. Transfer of a retroviral vector carrying the MDR-1 gene into bone marrow stem cells and their subsequent reintroduction will protect stem cells in vivo from the effects of large doses of taxol.

7. Genetic alteration of cancer cells in situ:

7.1. Liposome-mediated gene transfer : The genetic modification of tumors in situ involves the direct injection of liposomes containing an allogene that encoded HLA-B7, a foreign antigen that is transiently expressed on the cell surface and includes an immune reaction against the altered tumor cells. Anti-tumor immune response is significantly increased when some of the tumor cells express foreign antigens on their cell surface. The transient expression of immunostimulatory genes in tumors might have potential as a treatment and as a vaccination against certain malignancies.

7.2. Retrovirus-mediated gene transfer : In vivo gene transfer using murine retroviral vectors has been applied to the treatment of brain tumors. In this process, murine fibroblasts

that are actively producing retroviral vectors, so-called retro viral vector producer cells or VPCs, are implanted directly into growing tumors. The gene transferred by the retroviral vectors into the surrounding tumor cells is the HSVtk gene. The HSVtk gene should integrate only into the proliferating tumor cells because retrovirus-mediated gene transfer is limited to mitotically active cells. This technique resulted in transfer of the gene for HSVtk into 30-60% of brain tumor cells and was capable of mediating complete tumor destruction in 80% of patients. More than 50% of the cancers can be eliminated completely, At least 10% of cells in a tumor contain HSVtk, and adjacent tumor cells that do not contain HSVtk are destroyed through the bystander effect. No associated systemic toxicity or evidence of systemic spread of the retroviral vectors is seen with this form of in vivo gene transfer. So far, however, it is not clear whether this gene delivery system will suffice to eradicate the larger, infiltrative human tumors. Two protocols for in vivo gene transfer for cancer therapy have been approved for clinical trials. Both entail the direct injection of a supernatant containing a retroviral vector (RV) into tumor deposits. One group will inject two different RVs into endobronchial non-small-cell lung cancers. The vectors will carry genes that target the genetic mechanisms responsible for the malignancy: for example, if the lung tumors are deficient in expression of the P53 tumor suppressor gene, this gene will be used. In lung cancers that over express the K-RAS oncogene, a vector containing an antisense K-RAS gene will be used. Experiments in vitro have demonstrated that the introduction of both such vectors can result in decreased tumorigenicity. Another group will inject a RV containing a vector that encodes r-interferon directly into melanoma deposits.

There is an important distinction between somatic gene therapy (DNA transfer to our normal body tissue) and germ line gene therapy (DNA transfer to cells that produce eggs or sperm). The distinction is that the results of any somatic gene therapy are restricted to the actual patient and are not passed on to his or her children. There are some arguments in favour of germ line gene therapy (for example, it would allow the correction of disease-causing mutations that are certain to be passed on) but many more arguments against. The principle objections are:

- The technology is imperfect. The effects of gene transfer are unpredictable and, even if the target disease was cured, further defects could be introduced into the embryo.
- Denial of human rights. Individuals resulting from germ line gene therapy would have no say in whether their genetic material should have been modified.
- Potential abuse. Germ line gene therapy could be used not only to eliminate disease, but also to enhance favorable characteristics and suppress unfavorable ones. On a small scale, this would result in a generation of 'designer children', with traits chosen by their parents. On a large scale, gene therapy could result in eugenics - manipulation of the genetic properties of a population.

8. Technological hurdles or problems with the gene therapy:

Some of the unsolved problems with the technology underlying gene therapy include:

- **Short-lived nature of gene therapy** – Before gene therapy can become a permanent cure for any condition, the therapeutic DNA introduced into target cells must remain functional and the cells containing the therapeutic DNA must be long-lived and stable. Problems with integrating therapeutic DNA into the genome and the rapidly dividing nature of many cells prevent gene therapy from achieving any long-term benefits. Patients will have to undergo multiple rounds of gene therapy.
- **Immune response** – Any time a foreign object is introduced into human tissues; the immune system is stimulated to attack the invader. The risk of stimulating the immune system in a way that reduces gene therapy effectiveness is always a possibility. Furthermore, the immune system's enhanced response to invaders that it has seen before makes it difficult for gene therapy to be repeated in patients.
- **Problems with viral vectors** – Viruses, the carrier of choice in most gene therapy studies, present a variety of potential problems to the patient: toxicity, immune and inflammatory responses, and gene control and targeting issues. In addition, there is always the fear that the viral vector, once inside the patient, may recover its ability to cause disease.
- **Multi gene disorders** – Conditions or disorders that arise from mutations in a single gene are the best candidates for gene therapy. Unfortunately, some of the most commonly occurring disorders, such as heart disease, high blood pressure, Alzheimer's disease, arthritis, and diabetes, are caused by the combined effects of variations in many genes. Multi gene or multi factorial disorders such as these would be especially difficult to treat effectively using gene therapy.
- For countries in which germ-line gene therapy is illegal, indications that the Weismann barrier (between soma and germ-line) can be breached are relevant; spread to the testes, therefore could impact the germ line against the intentions of the therapy.
- **Chance of inducing a tumor (insertional mutagenesis)** – If the DNA is integrated in the wrong place in the genome, for example in a tumor suppressor gene, it could induce a tumor. This has occurred in clinical trials for X-linked severe combined immunodeficiency (X-SCID) patients, in which hematopoietic stem cells were transduced with a corrective transgene using a retrovirus, and this led to the development of T cell leukemia in 3 of 20 patients. One possible solution for this is to add a functional tumor suppressor gene onto the DNA to be integrated; however, this poses its own problems, since the longer the DNA is, the harder it is to integrate it efficiently into cell genomes. The development of CRISPR technology in 2012 allowed researchers to make much more precise changes at exact locations in the genome.
- **The cost** - only a small number of patients can be treated with gene therapy because of the extremely high cost (Alipogene tiparvovec or Glybera, for example, at a cost of \$1.6

million per patient was reported in 2013 to be the most expensive drug in the world).

9. Deaths:

Three patients' deaths have been reported in gene therapy trials, putting the field under close scrutiny. The first was that of Jesse Gelsinger in 1999, which represented a major setback in the field. One X-SCID patient died of leukemia following gene therapy treatment in 2003. In 2007, a rheumatoid arthritis patient died from an infection in a gene therapy trial; a subsequent investigation concluded that the death was not related to her gene therapy treatment.

10. Speculative uses for gene therapy:

Several uses for gene therapy have been speculated including:

10.1. Gene doping : There is a risk that athletes might abuse gene therapy technologies to improve their athletic performance. This idea is known as gene doping and is as yet not known to be in use but a number of gene therapies have potential applications to athletic enhancement. In some cases, scholars have argued that genetic technology can make doping safer and thus more ethically acceptable. In other cases, scientists and medics consider that any application of a therapeutic intervention for non-therapeutic or enhancing purposes compromises the ethical foundation of medicine and the spirit of sport.

10.2. Human genetic engineering : It has been speculated that genetic engineering could be used to change physical appearance, metabolism, and even improve physical capabilities and mental faculties like memory and intelligence, although for now these uses are limited to science fiction. These speculations have in turn led to ethical concerns and claims, including the belief that every fetus has an inherent right to remain genetically unmodified, the belief that parents hold the rights to modify their unborn offspring, and the belief that every child has the right to be born free from preventable diseases. On the other hand, others have made claims that many people try to improve themselves already through diet, exercise, education, cosmetics, and plastic surgery and that accomplishing these goals through genetics could be more efficient and worthwhile. This view sees the prevention of genetic diseases as a duty to humankind in preventing harm to future generations.

11. Regulations:

Policies on genetic modification tend to fall in the realm of general guidelines about human-involved biomedical research. Universal restrictions and documents have been made by international organizations to set a general standard on the issue of involving humans directly in research. One key regulation comes from the Declaration of Helsinki (Ethical Principles for Medical Research Involving Human Subjects), last amended by the World Medical Association's General Assembly in 2008. This document focuses on the principles physicians and researchers must consider when involving humans as the research subject. Additionally, the Statement on Gene Therapy Research initiated by the Human Genome Organization in 2001 also provides a legal baseline for all countries. HUGO's document

reiterates the organization's common principles researchers must follow when conducting human genetic research including the recognition of human freedom and adherence to human rights, and the statement also declares recommendations for somatic gene therapy including a call for researchers and governments to attend to public concerns about the pros, cons and ethical concerns about the research.

11.1. United States : No federal legislation specifically lays out protocol and restrictions about either germline or somatic human genetic engineering. Instead, this subject is governed by overlapping regulations from local and federal agencies. Included agencies, from the Department of Health and Human Services, are the Food and Drug Administration and the Recombinant DNA Advisory Committee of the National Institutes of Health. Additionally, researchers who wish to receive federal funds when conducting research about an investigational new drug application, which is commonly the case for somatic human genetic engineering, are required to obey international and federal guidelines dealing with the protection of human test subjects. The National Institutes of Health (NIH) mainly serves as the gene therapy regulator for federally funded research institutions and projects. Privately funded human genetic research can only be recommended to voluntarily follow their regulations. NIH provides funding for lab research that develops or enhances devices utilized in human genetic engineering and to evaluate the ethics and quality of science present in current research labs. The NIH maintains a mandatory registry of human genetic engineering research protocols from all federally funded projects. An advisory committee to the NIH published a set of guidelines on the manipulation of genes. The document for the NIH guidelines discusses safety considerations for the lab as well as for any human patient test subject. A wide range of various experimental types which involve any type of gene transfer or alteration are discussed. Several sections specifically pertain to human genetic engineering including Section III-C-1. This section states the review process researches must undergo and the aspects that are considered when attempting to be approved to begin clinical research involving human genetic transfer into a patient. This document is an important tool required for scientists to follow in order to further scientific progress in the field of somatic cell therapy. The United States Food and Drug Administration (FDA) regulate the quality and safety of gene therapy products and supervise how these products are implicated clinically. Therapeutic alteration of the human genome falls under the same regulatory requirements as any other medical treatment. Research involving human subjects, such as clinical trials, must be reviewed and approved by the FDA and an Institutional Review Board.

Gene therapy is under study to determine whether it could be used to treat disease. Current research is evaluating the safety of gene therapy; future studies will test whether it is an effective treatment option. Several studies have already shown that this approach can have very serious health risks, such as toxicity, inflammation, and cancer. Because the techniques are relatively new, some of the risks may be unpredictable; however, medical researchers, institutions, and regulatory agencies are working to ensure that gene therapy research is as

safe as possible.

The National Institutes of Health (NIH) plays an important role in ensuring the safety of gene therapy research. NIH provides guidelines for investigators and institutions (such as universities and hospitals) to follow when conducting clinical trials with gene therapy. These guidelines state that clinical trials at institutions receiving NIH funding for this type of research must be registered with the NIH Office of Biotechnology Activities. The protocol, or plan, for each clinical trial is then reviewed by the NIH Recombinant DNA Advisory Committee (RAC) to determine whether it raises medical, ethical, or safety issues that warrant further discussion at one of the RAC's public meetings. An Institutional Review Board (IRB) and an Institutional Bio safety Committee (IBC) must approve each gene therapy clinical trial before it can be carried out. An IRB is a committee of scientific and medical advisors and consumers that reviews all research within an institution. An IBC is a group that reviews and approves an institution's potentially hazardous research studies. Multiple levels of evaluation and oversight ensure that safety concerns are a top priority in the planning and carrying out of gene therapy research.

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STEM CELL THERAPY

Stem cells are very special, powerful cells found in both humans and non-human animals. They have been called the centerpieces of regenerative medicine that involves growing new cells, tissues and organs to replace or repair those damaged by injury, disease or aging. Stem cells are the precursors of all cells in the human body. They have the ability to replicate themselves and to repair and replace other tissues in the human body. Some tissues, like skin, need constant renewal, which could not take place without skin stem cells. Other stem cells repair damage to the body's tissues, for example, rebuilding damaged or degenerating muscle tissue. New research also indicates that stem cell malfunction or damage may be responsible for certain cancers and even muscular-degeneration diseases like muscular dystrophy. Research on stem cell functioning is therefore a critical avenue to finding treatments for these and other diseases. In the year of 1981, scientists discovered ways to derive embryonic stem cells from early mouse embryos. The detailed study of the biology of mouse stem cells led to the discovery, in 1998, of a method to derive stem cells from human embryos and grow the cells in the laboratory. These cells are called human embryonic stem cells. The embryos used in these studies were created for reproductive purposes through in vitro fertilization procedures. In 2006, researchers made another breakthrough by identifying conditions that would allow some specialized adult cells to be “reprogrammed” genetically to assume a stem cell-like state. Stem cells differ according to their source and their malleability. Commonly, stem cells come from two main sources:

- Adult tissue (adult stem cells).
- Embryos formed during the blastocyst phase of embryological development (embryonic stem cells).

Both types are generally characterized by their potency, or potential to differentiate into different cell types (such as skin, muscle, bone, etc.).

Adult stem cells : Adult or somatic stem cells exist throughout the body after embryonic development and are found inside of different types of tissue. These stem cells have been found in tissues such as the brain, bone marrow, blood, blood vessels, skeletal muscles, skin, and the liver. They remain in a quiescent or non-dividing state for years until activated by disease or tissue injury. Adult stem cells can divide or self-renew indefinitely, enabling them to generate a range of cell types from the originating organ or even regenerates the entire original organ. It is generally thought that adult stem cells are limited in their ability to differentiate based on their tissue of origin, but there is some evidence to suggest that they can differentiate to become other cell types.

Embryonic stem cells : Embryonic stem cells are derived from a four-or five-day-old human embryo that is in the blastocyst phase of development. The embryos are usually extras that have been created in IVF (in vitro fertilization) clinics where several eggs are fertilized in

a test tube, but only one is implanted into a woman. Sexual reproduction begins when a male's sperm fertilizes a female's ovum (egg) to form a single cell called a zygote. The single zygote cell then begins a series of divisions, forming 2, 4, 8, 16 cells, etc. After four to six days-before implantation in the uterus, this mass of cells is called a blastocyst. The blastocyst consists of an inner cell mass (embryoblast) and an outer cell mass (trophoblast). The outer cell mass becomes part of the placenta, and the inner cell mass is the group of cells that will differentiate to become all the structures of an adult organism. This latter mass is the source of embryonic stem cells commonly known as totipotent cells (cells with total potential to develop into any cell in the body).

In a normal pregnancy, the blastocyst stage continues until implantation of the embryo in the uterus, at which point the embryo is referred to as a fetus. This usually occurs by the end of the 10th week of gestation after all major organs of the body have been created. However, when extracting embryonic stem cells, the blastocyst stage signals when to isolate stem cells by placing the "inner cell mass" of the blastocyst into a culture dish containing a nutrient-rich broth. Lacking the necessary stimulation to differentiate, they begin to divide and replicate while maintaining their ability to become any cell type in the human body. Eventually, these undifferentiated cells can be stimulated to create specialized cells.

There is a continuing debate over the properties possessed by adult human and embryonic stem cells. Research into adult stem cells continues to evaluate their ability to differentiate which may be more limited than embryonic stem cells. Another difference between adult stem cells and embryonic stem cells is their behavior in culture. It is easier to get embryonic stem cells to replicate in culture and to keep those cells alive for a very long time, an attribute that is very useful in creating cell lines to study diseases or cell functioning. Regardless of their differences, both avenues of research will likely yield valuable and different information for the development of clinical therapies.

The specific factors and conditions that allow stem cells to remain unspecialized are of great interest to scientists. It has taken scientists many years of trial and error to learn to derive and maintain stem cells in the laboratory without them spontaneously differentiating into specific cell types. For example, it took two decades to learn how to grow human embryonic stem cells in the laboratory following the development of conditions for growing mouse stem cells. Likewise, scientists must first understand the signals that enable a non-embryonic (adult) stem cell population to proliferate and remain unspecialized before they will be able to grow large numbers of unspecialized adult stem cells in the laboratory. One of the fundamental properties of a stem cell is that it does not have any tissue-specific structures that allow it to perform specialized functions. For example, a stem cell cannot work with its neighbors to pump blood through the body (like a heart muscle cell), and it cannot carry oxygen molecules through the bloodstream (like a red blood cell). However, unspecialized stem cells can give rise to specialized cells, including heart muscle cells, blood cells or nerve cells.

When unspecialized stem cells give rise to specialized cells, the process is called

differentiation. While differentiating, the cell usually goes through several stages, becoming more specialized at each step. Scientists are just beginning to understand the signals inside and outside cells that trigger each step of the differentiation process. The internal signals are controlled by a cell's genes, which are interspersed across long strands of DNA and carry coded instructions for all cellular structures and functions. The external signals for cell differentiation include chemicals secreted by other cells, physical contact with neighboring cells, and certain molecules in the microenvironment. The interaction of signals during differentiation causes the cell's DNA to acquire epigenetic marks that restrict DNA expression in the cell and can be passed on through cell division.

Adult stem cells typically generate the cell types of the tissue in which they reside. For example, a blood-forming adult stem cell in the bone marrow normally gives rise to the many types of blood cells. It is generally accepted that a blood-forming cell in the bone marrow which is called a hematopoietic stem cell cannot give rise to the cells of a very different tissue, such as nerve cells in the brain. Experiments over the last several years have purported to show that stem cells from one tissue may give rise to cell types of a completely different tissue. This remains an area of great debate within the research community. This controversy demonstrates the challenges of studying adult stem cells and suggests that additional research using adult stem cells is necessary to understand their full potential as future therapies.

Important of stem cells:

Stem cells have the remarkable potential to develop into many different cell types in the body during early life and growth. In addition, in many tissues they serve as a sort of internal repair system, dividing essentially without limit to replenish other cells as long as the person or animal is still alive. When a stem cell divides, each new cell has the potential either to remain a stem cell or become another type of cell with a more specialized function, such as a muscle cell, a red blood cell, or a brain cell. Stem cells are distinguished from other cell types by two important characteristics. First, they are unspecialized cells capable of renewing themselves through cell division, sometimes after long periods of inactivity. Second, under certain physiologic or experimental conditions, they can be induced to become tissue or organ-specific cells with special functions. In some organs, such as the gut and bone marrow, stem cells regularly divide to repair and replace worn out or damaged tissues. In other organs, however, such as the pancreas and the heart, stem cells only divide under special conditions.

Stem cells are important for living organisms for many reasons. In the 3 to 5 day old embryo, called a blastocyst, the inner cells give rise to the entire body of the organism, including all of the many specialized cell types and organs such as the heart, lungs, skin, sperm, eggs and other tissues. In some adult tissues, such as bone marrow, muscle, and brain, discrete populations of adult stem cells generate replacements for cells that are lost through normal wear and tear, injury, or disease. Given their unique regenerative abilities, stem cells offer new potentials for treating diseases such as diabetes and heart disease. Laboratory studies of stem cells enable scientists to learn about the cells' essential properties and what

makes them different from specialized cell types. Scientists are already using stem cells in the laboratory to screen new drugs and to develop model systems to study normal growth and identify the causes of birth defects.

Research on stem cells continues to advance knowledge about how an organism develops from a single cell and how healthy cells replace damaged cells in adult organisms. Stem cell research is one of the most fascinating areas of contemporary biology, but, as with many expanding fields of scientific inquiry, research on stem cells raises scientific questions as rapidly as it generates new discoveries.

Sources of stem cells for transplant:

There are 3 possible sources of stem cells to use for transplants:

- Bone marrow
- The bloodstream (peripheral blood)
- Umbilical cord blood from newborns

Bone marrow : Bone marrow is the spongy tissue in the center of some bones. Its main job is to make blood cells that circulate in the body, which includes immune cells that recognize invaders and fight infection. Bone marrow has a rich supply of stem cells. The bones of the pelvis (hip) contain the most marrow and have large numbers of stem cells in them. For this reason, cells from the pelvic bone are used most often for a bone marrow transplant. Enough marrow must be removed to collect a large number of healthy stem cells. When the bone marrow is removed (harvested), the donor gets general anesthesia. A large needle is put through the skin and into the back of the hip bone. The thick liquid marrow is pulled out through the needle. This is repeated several times until enough marrow has been taken out or harvested. The harvested marrow is filtered, stored in a special solution in bags, and then frozen. When the marrow is to be used, it's thawed and then given into the vein just like a blood transfusion. The stem cells travel to the recipient's bone marrow. Over time, they engraft or "take" and begin to make blood cells. Signs of the new blood cells usually can be measured in the patient's blood tests in about 2 to 4 weeks.

Peripheral blood : Normally, few stem cells are found in the blood. But giving hormone-like substances called growth factors to stem cell donors a few days before the harvest causes their stem cells to grow faster and move from the bone marrow into the blood. For a peripheral blood stem cell transplant, the stem cells are taken from blood. A special thin flexible tube (called a catheter) is put into a large vein in the donor and attached to tubing that carries the blood to a special machine. The machine separates the stem cells from the rest of the blood, which is given back to the donor during the same procedure. This takes several hours, and may need to be repeated for a few days to get enough stem cells. The stem cells are filtered, stored in bags, and frozen until the patient is ready for them. After the patient is treated with chemotherapy and/or radiation, the stem cells are infused into the vein, much like a blood transfusion. The stem cells travel to the bone marrow, engraft and then start making new,

normal blood cells. The new cells are usually found in the patient's blood a few days sooner than when bone marrow stem cells are used, usually in about 10 to 20 days.

Umbilical cord blood : The first cord blood transplant was done in 1988, and its use has been growing ever since. Not everyone who needs an allogeneic stem cell transplant can find a well-matched donor among family members or among the people who have signed up to donate. For these patients, umbilical cord blood may be a source of stem cells. About 1 in 3 unrelated hematopoietic stem cell transplants are done with cord blood. A large number of stem cells are normally found in the blood of newborn babies. After birth, the blood that is left behind in the placenta and umbilical cord (known as cord blood) can be taken and stored for later use in a stem cell transplant. The cord blood is frozen until needed. A cord blood transplant uses blood that normally is thrown out after a baby is born. A possible drawback of cord blood is the smaller number of stem cells present. But this is partly balanced by the fact that each cord blood stem cell can form more blood cells than a stem cell from adult bone marrow. Still, cord blood transplants can take longer to take hold and start working. To be safe, most cord blood transplants done so far have been in children and smaller adults. Researchers are now looking for ways to use cord blood for transplants in larger adults. One approach that is being taken is to find ways to increase the numbers of these cells in the lab before the transplant. Another approach is the use of the cord blood from 2 infants at the same time for one adult transplant, called a dual-cord-blood transplant. A third way cord blood is being used is in a mini-transplant. In this case, the bone marrow is not completely destroyed so there are some host stem cells left before and during the time that the cord blood stem cells engraft. Other strategies to better use cord blood transplants are being actively studied.

All 3 sources of stem cells can be used for the same goal : to give the patient healthy stem cells that will mature into healthy blood cells. There are pros and cons to each source, but all are usually able to provide the needed number of stem cells. When stem cell transplants were first used, they were all bone marrow transplants. But today peripheral blood stem cell transplants are much more common. Often, doctors are able to harvest more stem cells from peripheral blood than from bone marrow. It's also easier for donors to give peripheral blood stem cells than bone marrow, although it takes longer. Another plus for peripheral blood stem cell transplants is that the recipient's blood count often recovers faster than a bone marrow transplant. But the risk of chronic graft-versus-host disease is somewhat higher with peripheral blood stem cell transplants than with bone marrow transplants.

Cord blood transplant may be an option if a good match can't be found among volunteer stem cell donors. Even though well-matched cord blood is generally best, studies suggest that cord blood doesn't have to be as closely matched as bone marrow or peripheral blood. This may be an advantage for patients with rare tissue types. This type of transplant also does not require a separate donation procedure and may reduce the risk and severity of graft-versus-host disease. But cord blood cells usually take longer to engraft. This leaves the patient at high risk for infection and bleeding longer than is seen with transplanted marrow or peripheral

blood stem cells. Another drawback is that, unlike bone marrow transplant or peripheral blood stem cell transplant, the donor cannot be called back for more after the cord blood stem cells are used.

Stem cells and cancer:

Ideally, embryonic stem cells (ESCs) would be the source of stem cells for therapeutic purposes due to higher totipotency and indefinite life span compared to adult stem cells (ASCs) with lower totipotency and restricted life span. However, uses of ESCs have ethical constraints (Department of Health, UK, National Institutes of Health and International Society for Stem Cell Research) and their use for research and therapeutic purposes are restricted and prohibited in many countries throughout the world. In addition, the stem cells with higher totipotency have been shown to be more tumorigenic in mice. Thus, for ease of availability and lesser constrained on ethical issue, ASCs are the stem cells most commonly used for research and therapeutic purposes. The other reason for the use of ASCs is their easy accessibility compared to ESCs. According to literature, ASCs from bone marrow of haemopoietic stem cells (HSCs) and mesenchymal stem cells (MSCs) are the most commonly studied stem cells. MSCs support HSCs in the bone marrow and have the ability to differentiate both in vivo and in vitro into the different mesenchymal cells such as bone, cartilage, fat, muscle, tendon and marrow stroma.

All of the blood cells in body start out as young (immature) cells called hematopoietic stem cells. Even though they are called stem cells, they are not the same as the embryos' stem cells that are studied in cloning and other types of research. The words stem cells refer to blood-forming stem cells. Stem cells mostly live in the bone marrow (the spongy center of certain bones), where they divide to make new blood cells. Once blood cells mature, they leave the bone marrow and enter the bloodstream. A small number of stem cells also get into the bloodstream. These are called peripheral blood stem cells.

Stem cell transplants are used to restore the stem cells when the bone marrow has been destroyed by disease, chemotherapy (chemo), or radiation. Depending on the source of the stem cells, this procedure may be called a bone marrow transplant, a peripheral blood stem cell transplant, or a cord blood transplant. They can all be called hematopoietic stem cell transplants. Today thousands of patients have had stem cell transplants. Transplant teams are better able to care for transplant patients and doctors know more about which patients are likely to have better results after transplant. Stem cells make the 3 main types of blood cells: red blood cells, white blood cells, and platelets. We need all of these types of blood cells to keep us alive.

Red blood cells (erythrocytes) : Red blood cells (RBCs) carry oxygen from the lungs to all of the cells in the body, and then bring carbon dioxide back from the cells to the lungs to be exhaled. A blood test called a hematocrit shows how much blood is made up of RBCs. The normal range is about 35% to 50% for adults. People whose hematocrit is below this level have anemia. This can make them look pale and feel weak, tired, and short of breath.

White blood cells (leukocytes) : White blood cells (WBCs) are a crucial part of the immune system. They help fight infections caused by bacteria, viruses and fungi. There are different types of WBCs. Neutrophils are the most important type in fighting bacterial infections. The absolute neutrophil count (ANC) is a measure of the neutrophils in blood. When ANC drops below 1,000 per cubic millimeter (1,000/mm³) have neutropenia, and have a higher risk of serious infection. The danger is greatest when levels are below 500/mm³. Stem cells make another type of white blood cell called lymphocytes. There are different kinds of lymphocytes, such as T lymphocytes (T cells), B lymphocytes (B cells), and natural killer (NK) cells. Some lymphocytes make antibodies to help fight infections. The body depends on lymphocytes to recognize its own cells and reject cells that don't belong to the body, such as invading germs or cells that are transplanted from someone else.

Platelets (thrombocytes) : Platelets are pieces of cells that seal damaged blood vessels and help blood clot, both of which are important in stopping bleeding. A normal platelet count is usually between 150,000/cubic mm and 450,000/cubic mm, depending on the lab that does the test. A person whose platelet count drops below normal is said to have thrombocytopenia, and may bruise more easily, bleed longer, and have nosebleeds or bleeding gums. Spontaneous bleeding (bleeding with no known injury) can happen if a person's platelet count drops lower than 20,000/mm³. This can be dangerous if bleeding occurs in the brain, or if blood begins to leak into the intestine or stomach.

Stem cells in immune reconstitution :

The stem cells have been used since many years in immuno-reconstitution following cancer development or following cancer treatments. The high dose chemotherapy has the adverse effects on the bone marrow causing myelosuppression. Usually this is followed by the blood cell recovery through the haematopoietic progenitor cells residing in the bone marrow by the complex interactions between the progenitor cells and the marrow microenvironment under the influence of various stimulatory and inhibitory factors. However, time for haematopoietic recovery is proportional to the doses and number of cycles of chemotherapy. It has been shown that chemotherapy can induce inhibitory factors such as Tumour Growth Factor (TGF)- α , Interferon (IFN)- α – IFN- γ , Tumour Necrosis Factor(TNF)- α and Interleukin(IL)-4 with cytokines that causes myelosuppression. HSCs are the most commonly used and they are the stem cells of choice for the haematopoietic cell transplantation following high dose chemotherapy to restore bone marrow and immune system to pre-chemotherapy levels. Randomised clinical trials regarding the use of HSCs for haematopoietic cell transplantation have been published with controversial results, however most of the trials suggested improved disease free survival rates, shorter hospital stay, overall survival rates and event free survival rates, while fewer of the studies have reported no statistically significant differences when assessed those parameters. The adequate number of the stem cells therapy is also reported a crucial factors for speedy recovery. Some of the chemotherapeutic agents, especially alkylating agents, should be avoided as they are reported to adversely affect stem

cell yield and haematopoietic recovery. The post-transplant period thrombocytopenia and neutropenia may be reduced by re-infusion of ex vivo expanded mega caryocyte progenitors and re-infusion of ex vivo expanded peripheral blood stem cells (PBSC) respectively.

Choice of type of stem cell : The source of stem cells is most commonly either from the bone marrow or the peripheral blood. The procedure of the bone marrow aspiration is invasive and is associated with the potential possible complications including fracture, wound infection and sepsis while the procedure for PBSCs isolation is much less invasive and less morbid. PBSCs have also been shown to induce higher number of CD4 T and NK cells compared to stem cells obtained from the bone marrow. Thus, the stem cells from peripheral blood are considered the preferred source of stem cells however various clinical trials have publicized controversial conclusions comparing PBSCs and BM stem cells. It is also noticed that the occurrence of graft versus host reaction varies with PBSCs compared to BM stem cells. Clinical trials provide different outcomes such as PBSC yields higher lymphocyte subset counts while no difference in the number of lymphocyte counts but noted faster reconstitution of cytotoxic subsets. Similarly, these trials present controversial results including graft versus host disease, overall survival, disease free survival and immune recovery. Double stem cell transplantation has been documented to improve overall survival compared to single stem cell transplantation. Granulocyte-colony stimulating factor (G-CSF) helps in proliferation and differentiation of haematopoietic progenitor cells. G-CSF has also been reported to mobilise autologous peripheral blood stem cells and to preserve and increase the length of telomerase. There are various different agents which are shown to enhance the G-CSG activity in mobilizing stem cell. These are paclitaxel and docetaxel, recombinant human thrombopoietin, lithium and recombinant methionyl human stem cell factor (r-metHuSCF).

Stem cell transplantation : Stem cell transplants are used to replace bone marrow that isn't working or has been destroyed by disease, chemo, or radiation. In some diseases, like leukemia, aplastic anemia, certain inherited blood diseases, and some diseases of the immune system, the stem cells in the bone marrow don't work the way they should. Damaged or diseased stem cells can make too few blood cells, too few immune cells, or too many abnormal cells. Any of these problems can cause the body to not have enough normal red blood cells, white blood cells, or platelets. A stem cell transplant may help correct these problems. In some cancers, such as certain leukemias, multiple myeloma, and some lymphomas, a stem cell transplant can be an important part of treatment. It works like, high doses of chemo, which is sometimes given with radiation; work well than standard doses to kill cancer cells. But high doses can also cause the bone marrow to completely stop making blood cells, which we need to live. This is where stem cell transplants come in. The transplanted cells replace the body's source of blood cells after the bone marrow and its stem cells have been destroyed by the treatment. This transplant lets doctors use much higher doses of chemo to try to kill all of the cancer cells.

A stem cell transplant from another person can also help treat certain types of cancer

in a way other than just replacing stem cells. Donated cells can often find and kill cancer cells better than the immune cells of the person who had the cancer ever could. This is called the “graft-versus-cancer” or “graft-versus-leukemia” effect. It means that certain kinds of transplants actually help fight the cancer cells, rather than simply replacing the blood cells.

Stem cell transplants and cancer treatment:

In a typical stem cell transplant for cancer very high doses of chemo are used, often along with radiation therapy, to try to destroy all the cancer cells. This treatment also kills the stem cells in the bone marrow. Soon after treatment, stem cells are given to replace those that were destroyed. These stem cells are given into a vein, much like a blood transfusion. Over time they settle in the bone marrow and begin to grow and make healthy blood cells. This process is called engraftment. There are 3 basic types of transplants. They are named based on who gives the stem cells.

- Autologous -the cells come from him.
- Allogeneic - the cells come from a matched related or unrelated donor.
- Syngeneic - the cells come from identical twin or triplet

Autologous stem cell transplants : In this type of transplant, stem cells are taken before get cancer treatment that destroys them. Stem cells are removed, or harvested, from either bone marrow or blood and then frozen. One advantage of autologous stem cell transplant is that you are getting your own cells back. When you donate your own stem cells you don't have to worry about the graft attacking your body (graft-versus-host disease) or about getting a new infection from another person. But there can still be graft failure, and autologous transplants can't produce the “graft-versus-cancer” effect.

This kind of transplant is mainly used to treat certain leukemias, lymphomas, and multiple myeloma. It's sometimes used for other cancers, like testicular cancer and neuroblastoma, and certain cancers in children. Doctors are looking at how autologous transplants might be used to treat other diseases, too, like systemic sclerosis, multiple sclerosis, Crohn disease, and systemic lupus erythematosus.

Cancer cells in autologous transplants : A possible disadvantage of an autologous transplant is that cancer cells may be picked up along with the stem cells and then put back into the body. Another disadvantage is that immune system is still the same as before when stem cells engraft. The cancer cells were able to grow despite our immune cells before, and may be able to do so again. To prevent this, doctors may give anti-cancer drugs or treat stem cells in other ways to reduce the number of cancer cells that may be present. Some centers treat the stem cells to try to remove any cancer cells before they are given back to the patient. This is sometimes called “purging.” It isn't clear that this really helps, as it has not yet been proven to reduce the risk of cancer coming back (recurrence). A possible downside of purging is that some normal stem cells can be lost during this process, causing the patient to take longer to begin making normal blood cells, and have unsafe levels of white blood cells or platelets for

a longer time. This could increase the risk of infections or bleeding problems. One popular method now is to give the stem cells without treating them. Then, after transplant, the patient gets a medicine to get rid of cancer cells that may be in the body. This is called in vivo purging. Rituximab (Rituxan®), a monoclonal antibody drug, may be used for this in certain lymphomas and leukemias, and other drugs are being tested. The need to remove cancer cells from transplants or transplant patients and the best way to do it is being researched.

Tandem transplants : Doing 2 autologous transplants in a row is known as a tandem transplant or a double autologous transplant. In this type of transplant, the patient gets 2 courses of high-dose chemo, each followed by a transplant of their own stem cells. All of the stem cells needed are collected before the first high-dose chemo treatment, and half of them are used for each transplant. Most often both courses of chemo are given within 6 months, with the second one given after the patient recovers from the first one. Tandem transplants are most often used to treat multiple myeloma and advanced testicular cancer, but doctors do not always agree that these are really better than a single transplant for certain cancers. Because this involves 2 transplants, the risk of serious outcomes is higher than for a single transplant. Tandem transplants are still being studied to find out when they might be best used. Sometimes an autologous transplant followed by an allogeneic transplant might also be called a tandem transplant.

Allogeneic stem cell transplants : In the most common type of allogeneic transplant, the stem cells come from a donor whose tissue type closely matches the patient's. The best donor is a close family member, usually a brother or sister. If patient do not have a good match in family, a donor might be found in the general public through a national registry. This is sometimes called a MUD (matched unrelated donor) transplant. Transplants with a MUD are usually riskier than those with a relative who is a good match. Blood taken from the placenta and umbilical cord of newborns is a newer source of stem cells for allogeneic transplant. Called cord blood, this small volume of blood has a high number of stem cells that tend to multiply quickly. But the number of stem cells in a unit of cord blood is often too low for large adults, so this source of stem cells is limited to small adults and children. Doctors are now looking at different ways to use cord blood for transplant in larger adults, such as using cord blood from 2 donors.

Process of allogeneic stem cell transplant : The donor stem cells make their own immune cells, which could help destroy any cancer cells that remain after high-dose treatment. This is called the graft-versus-cancer effect. Other advantages are that the donor can often be asked to donate more stem cells or even white blood cells if needed, and stem cells from healthy donors are free of cancer cells. The transplant, also known as the graft, might not take—that is, the donor cells could die or be destroyed by the patient's body before settling in the bone marrow. Another risk is that the immune cells from the donor may not just attack the cancer cells—they could attack healthy cells in the patient's body. This is called graft-versus-host disease. There is also a very small risk of certain infections from the donor cells, even though

donors are tested before they donate. A higher risk comes from infections and which immune system has under control. These infections often surface after allogeneic transplant because immune system is held in check (suppressed) by medicines called immunosuppressive drugs. These infections can cause serious problems and even death. Allogeneic transplant is most often used to treat certain types of leukemia, lymphomas, multiple myeloma, myelodysplastic syndrome, and other bone marrow disorders such as aplastic anemia.

Mini transplants (non-myeloablative transplants) : For some people, age or certain health conditions make it more risky to wipe out all of their bone marrow before a transplant. For those people, doctors can use a type of allogeneic transplant that's sometimes called a mini-transplant. Compared with a standard allogeneic transplant, this one uses less chemo and/or radiation to get the patient ready for the transplant. Doctor might refer to it as a non-myeloablative transplant or mention reduced-intensity conditioning (RIC). The idea here is to kill some of the cancer cells along with some of the bone marrow, and suppress the immune system just enough to allow donor stem cells to settle in the bone marrow. Unlike the standard allogeneic transplant, cells from both the donor and the patient exist together in the patient's body for some time after a mini-transplant. But slowly, over the course of months, the donor cells take over the bone marrow and replace the patient's own bone marrow cells. These new cells can then develop an immune response to the cancer and help kill off the patient's cancer cells — the graft-versus-cancer effect.

One advantage of a mini-transplant is the lower doses of chemotherapy (chemo) and/or radiation. And because the stem cells aren't all killed, blood cell counts don't drop as low while waiting for the new stem cells to start making normal blood cells. This makes it especially useful in older patients and those with other health problems who aren't strong enough for a standard allogeneic stem cell transplant. Rarely, it may be used in patients who have already had a transplant. Mini-transplants treat some diseases better than others. They may not work well for patients with a lot of cancer in their body or those with fast-growing cancers. Also, although side effects from chemo and radiation may be less than those from a standard allogeneic transplant, the risk of graft-versus-host disease is not. This procedure has only been used since the late 1990s and long-term patient outcomes are not yet clear. There are lower risks of some complications, but the cancer may be more likely to relapse (come back). Ways to improve outcomes are still being studied. Studies have looked at using an allogeneic mini-transplant after an autologous transplant. This is another type of tandem transplant being tested in certain types of cancer, such as multiple myeloma. The autologous transplant can help decrease the amount of cancer present so that the lower doses of chemo given before the mini-transplant can work better. And the recipient still gets the benefit of the graft-versus-cancer effect of the allogeneic transplant.

Syngeneic stem cell transplants—for those with an identical sibling : This is a special kind of allogeneic transplant that can only be used when the recipient has an identical sibling (twin or triplet) who can donate—someone who will have the same tissue type. An advantage

of syngeneic stem cell transplant is that graft-versus-host disease will not be a problem. There are no cancer cells in the transplant, either, as there would be in an autologous transplant.

A disadvantage is that because the new immune system is so much like the recipient's immune system, there is no graft-versus-cancer effect, either. Every effort must be made to destroy all the cancer cells before the transplant is done to help keep the cancer from relapsing (coming back).

Half-matched transplants : Some centers are doing half-match (haploidentical) transplants for people who don't have closely matching family members. This technique is most often used in children, usually with a parent as the donor, though a child can also donate to a parent. Half of the HLA factors will match perfectly, and the other half typically don't match at all, so the procedure requires a special way to get rid of a certain white blood cells that can cause graft-versus-host disease. It's still rarely done, but it's being studied in a few centers in the United States. Researchers are continuing to learn new ways to make haploidentical transplants more successful.

Donor matching for allogeneic transplant : The immune system normally keeps us healthy by destroying anything in the body it sees as foreign, such as bacteria or viruses. A working immune system recognizes cells from other people as foreign, too. This becomes very important in an allogeneic stem cell transplant. If the tissue type match between donor and recipient is not close, the patient's immune system may see the new stem cells as foreign and destroy them. This is called graft rejection, and it can lead to graft failure. This is rare when the donor and recipient are well matched. A more common problem is that when the donor stem cells make their own immune cells, the new cells may see the patient's cells as foreign and turn against their new home. This type of attack is called graft-versus-host disease. The grafted stem cells attack the body of the person who got the transplant. This is another reason it's so important to find the closest match possible.

HLA matching : Many factors play a role in how the immune system knows the difference between self and non-self, but the most important for transplants is the human leukocyte antigen (HLA) system. Human leukocyte antigens are proteins found on the surface of most cells. They make up a person's tissue type, which is different from a person's blood type. Each person has a number of pairs of HLA antigens. We inherit one of each of these pairs from each of our parents (and pass one of each pair on to each of our children). Doctors try to match these antigens when finding a donor for a person getting a stem cell transplant. How well the donor's and recipient's HLA tissue types match plays a large part in whether the transplant will work. A match is better when all 6 of the known major HLA antigens are the same a 6 out of 6 match. People with these matches have a lower chance of graft-versus-host disease, graft rejection, having a weak immune system, and getting serious infections. For bone marrow and peripheral blood stem cell transplants, sometimes a donor with a single mismatched antigen is used a 5 out of 6 match. For cord blood transplants a perfect HLA match doesn't seem to be as crucial for success, and even a sample with a couple

of mismatched proteins may be OK.

Doctors keep learning more about better ways to match donors. Today, fewer tests may be needed, for siblings since their cells vary less than an unrelated donor. But more than the basic 6 HLA antigens are often tested on unrelated donors to reduce the risks of mismatched types. Sometimes doctors will want to look at 5 pairs of antigens, for example, to try and get a 10 out of 10 match. Certain transplant centers now require high-resolution matching, which looks more deeply into tissue types. Other centers are doing clinical trials with related half-matched donors and different chemotherapy schedules. This is an active area of research because it's often hard to find a good HLA match.

Finding a match : There are thousands of different combinations of possible HLA tissue types, which can make it hard to find an exact match. HLA antigens are inherited from both parents. If possible, the search for a donor usually starts with the patient's brothers and sisters (siblings), who have the same parents as the patient. The chance that any one sibling would be a perfect match is 1 out of 4.

If a sibling is not a good match, the search could then move on to relatives who are less likely to be a good match — parents, half siblings, and extended family, such as aunts, uncles, or cousins. (Spouses are no more likely to be good matches than other people who are not related.) If no relatives are found to be a close match, the transplant team will widen the search to the general public. As unlikely as it seems, it's possible to find a good match with a stranger. To help with this process, the team will use transplant registries. Registries serve as matchmakers between patients and volunteer donors. They can search for and access millions of possible donors and hundreds of thousands of cord blood units. The largest registry in the United States is Be the Match (formerly called the National Marrow Donor Program) which has recently merged with another agency, the Caitlin Raymond International Registry. They have access to millions of international records, and have successfully matched thousands of donors and recipients.

The chances of finding an unrelated donor match improve each year, as more volunteers sign up. Today, about half of white people who need a stem cell transplant may find a perfect match among unrelated donors. This drops to about 1 out of 10 people in other ethnic groups, mostly because their HLA types are more diverse and they seem to be less likely to take part in donor registries. Depending on a person's tissue typing, several other international registries also are available. Sometimes the best matches are found in people with a similar racial or ethnic background. Finding an unrelated donor can take months, though cord blood may be a little faster. A single match can require going through millions of records.

Now that transplant centers are more often using high-resolution tests, matching is becoming more complex. Perfect 10 out of 10 matches at that level are much harder to find. But transplant teams are also getting better at figuring out what kinds of mismatches they can get away with in which situations – that is, which mismatched sites are less likely to affect transplant success and survival. Keep in mind that there are stages to this process –

there may be several matches that look promising but don't work out as hoped. The team and registry will keep looking for the best possible match for you. If your team finds an adult donor through a transplant registry, the registry will contact the donor to set up the final testing and donation. If your team finds matching cord blood, the registry will have the cord blood sent to your transplant center.

Despite the possible short-term problems and those that can crop up after a while, stem cell transplant has been used to cure thousands of people with otherwise deadly cancers. Still, the possible risks and complications can threaten life, too. The expected risks and benefits must be weighed carefully before transplant. Research today is being done to not only to cure cancer, but also to improve transplant methods and reduce the risks.

CELL CULTURE

An important aspect of any biotechnological processes is the culture of animal cells in artificial media. These animal cells in culture are used in recombinant DNA technology, genetic manipulations and in a variety of industrial processes. Now-a-days it has become possible to use the cell and tissue culture in the areas of research which have a potential for economic value and commercialization. The animal cell cultures are being extensively used in production of vaccines, monoclonal antibodies, pharmaceutical drugs, cancer research, genetic manipulations etc. Animal cells e.g. egg cells are used for multiplication of superior livestock using a variety of techniques like cloning of superior embryonic cells, transformation of cultured cells leading to the production of transgenic animals. The animal cells are also used in vitro fertilization and transfer of embryos to surrogate mothers. Hence the establishment and maintenance of a proper animal culture is the first step towards using them as tools for biotechnology.

Cell culture is the complex process by which cells are grown under controlled conditions, generally outside of their natural environment. In practice, the term “cell culture” now refers to the culturing of cells derived from multi-cellular eukaryotes, especially animal cells. However, there are also cultures of plants, fungi, insects and microbes, including viruses, bacteria and protists. The historical development and methods of cell culture are closely interrelated to those of tissue culture and organ culture.

History:

The 19th-century English physiologist Sydney Ringer developed salt solutions containing chlorides of sodium, potassium, calcium and magnesium suitable for maintaining the beating of an isolated animal heart outside of the body. In 1885, Wilhelm Roux removed a portion of the medullary plate of an embryonic chicken and maintained it in a warm saline solution for several days, establishing the principle of tissue culture. Ross Granville Harrison, working at Johns Hopkins Medical School and then at Yale University, working in the period between 1907 to 1910, establishing the methodology of tissue culture. Cell culture techniques were advanced significantly in the 1940s and 1950s to support research in virology. Growing viruses in cell cultures allowed preparation of purified viruses for the manufacture of vaccines. The injectable polio vaccine developed by Jonas Salk was one of the first products mass-produced using cell culture techniques. This vaccine was made possible by the cell culture research of John Franklin Enders, Thomas Huckle Weller, and Frederick Chapman Robbins, who were awarded a Nobel Prize for their discovery of a method of growing the virus in monkey kidney cell cultures.

Historical events in the development of cell culture

- 1878: Claude Bernard proposed that physiological systems of an organism can be maintained in a living system after the death of an organism.

- 1885: Roux maintained embryonic chick cells in a saline culture.
- 1897: Loeb demonstrated the survival of cells isolated from blood and connective tissue in serum and plasma.
- 1903: Jolly observed cell division of salamander leucocytes in vitro.
- 1907: Harrison cultivated frog nerve cells in a lymph clot held by the 'hanging drop' method and observed the growth of nerve fibers in vitro for several weeks. He was considered by some as the father of cell culture.
- 1910: Burrows succeeded in long term cultivation of chicken embryo cell in plasma clots. He made detailed observation of mitosis.
- 1911: Lewis and Lewis made the first liquid media consisted of sea water, serum, embryo extract, salts and peptones. They observed limited monolayer growth.
- 1913: Carrel introduced strict aseptic techniques so that cells could be cultured for long periods.
- 1916: Rous and Jones introduced proteolytic enzyme trypsin for the subculture of adherent cells.
- 1923: Carrel and Baker developed 'Carrel' or T-flask as the first specifically designed cell culture vessel. They employed microscopic evaluation of cells in culture.
- 1927: Carrel and Rivera produced the first viral vaccine - Vaccinia.
- 1933: Gey developed the roller tube technique
- 1940s: The use of the antibiotics penicillin and streptomycin in culture medium decreased the problem of contamination in cell culture.
- 1948: Earle isolated mouse L fibroblasts which formed clones from single cells. Fischer developed a chemically defined medium, CMRL 1066.
- 1952: Gey established a continuous cell line from a human cervical carcinoma known as HeLa (Helen Lane) cells. Dulbecco developed plaque assay for animal viruses using confluent monolayers of cultured cells.
- 1954: Abercrombie observed contact inhibition: motility of diploid cells in monolayer culture ceases when contact is made with adjacent cells.
- 1955: Eagle studied the nutrient requirements of selected cells in culture and established the first widely used chemically defined medium.
- 1961: Hayflick and Moorhead isolated human fibroblasts (WI-38) and showed that they have a finite lifespan in culture.
- 1964: Littlefield introduced the HAT medium for cell selection.
- 1965: Ham introduced the first serum-free medium which was able to support the growth of some cells.
- 1965: Harris and Watkins were able to fuse human and mouse cells by the use of a virus.
- 1975: Kohler and Milstein produced the first hybridoma capable of secreting a

monoclonal antibody.

- 1978: Sato established the basis for the development of serum-free media from cocktails of hormones and growth factors.
- 1982: Human insulin became the first recombinant protein to be licensed as a therapeutic agent.
- 1985: Human growth hormone produced from recombinant bacteria was accepted for therapeutic use.
- 1986: Lymphoblastoid α IFN licensed.
- 1987: Tissue-type plasminogen activator (tPA) from recombinant animal cells became commercially available.
- 1989: Recombinant erythropoietin in trial.
- 1990: Recombinant products in clinical trial (HBsAG, factor VIII, HIVgp120, CD4, GM-CSF, EGF, mAbs, IL-2).

History Of Animal Cell Culture:

It was Jolly showed for the first time that the cells can survive and divide in vitro in the year of 1903. Ross Harrison, (1907) was able to show the development of nerve fibres from frog embryo tissue, cultured in a blood clot. Later, Alexis Carrel (1912) used tissue and embryo extracts as cultural media to keep the fragments of chick embryo heart alive. In the late 1940s, Enders, Weller and Robbins grew poliomyelitis virus in culture which paved way for testing many chemicals and antibiotics that affect multiplication of virus in living host cells. The significance of animal cell culture was increased when viruses were used to produce vaccines on animal cell cultures in late 1940s. For about 50 years, mainly tissue explants rather than cells were used for culture techniques, although later after 1950s, mainly dispersed cells in culture were utilized. In 1966, Alec Issacs discovered Interferon by infecting cells in tissue culture with viruses. He took filtrates from virus infected cells and grew fresh cells in the filtered medium. When the virus was reintroduced in the medium, the cells did not get infected. He proposed that cells infected with the virus secreted a molecule which coated onto uninfected cells and interfered with the viral entry. This molecule was called "Interferon". Chinese Hamster Ovary (CHO) cell lines were developed during 1980s. Recombinant erythropoietin was produced on CHO cell lines by AMGEN (U.S.A.). It is used to prevent anaemia in patients with kidney failure who require dialysis. After this discovery, the Food and Drug Administration (U.S.A) granted the approval for manufacturing erythropoietin on CHO cell lines. In 1982, Thilly and co-workers used the conventional conditions of medium, serum, and O₂ with suitable beads as carriers and grew certain mammalian cell lines to densities as high as 5×10^6 cells/ml. A lot of progress has been also made in the area of stem cell technology which will have their use in the possible replacement of damaged and dead cells. In 1996, Wilmut and co-workers successfully produced a transgenic sheep named Dolly through nuclear transfer technique. Thereafter, many such animals (like sheep, goat, pigs, fishes, birds etc.) were produced.

Recently in 2002, Clonaid, a human genome society of France claimed to produce a cloned human baby named EVE. For animals, if the explant maintains its structure and function in culture it is called as an 'organotypic culture'. If the cells in culture reassociate to create a three dimensional structure irrespective of the tissue from which it was derived, it is described as a 'histotypic culture'.

Salient Features of Animal cell culture

- Animal cells can grow in simple glass or plastic containers in nutritive media but they grow only to limited generations.
- Animal cells exhibit contact inhibition. In culture the cancer cells apparently differ from the normal cells. Due to uncontrolled growth and more rounded shape, they lose contact inhibition and pile over each other.
- c) There is a difference in the in vitro and in vivo growth pattern of cells.

For example

- ✓ there is an absence of cell-cell interaction and cell matrix interaction,
- ✓ there is a lack of three-dimensional architectural appearance, and
- ✓ changed hormonal and nutritional environment. Their way of adherence to glass or plastic container in which they grow, cell proliferation and shape of cell results in alterations.
- The maintenance of growth of cells under laboratory conditions in suitable culture medium is known as primary cell culture.
- Cells are dissociated from tissues by mechanical means and by enzymatic digestion using proteolytic enzymes.
- Cells can grow as adherent cells (anchorage dependent) or as suspension cultures (anchorage independent).
- The primary culture is subculture in fresh media to establish secondary cultures.
- The various types of cell lines are categorized into two types as Finite cell line and Continuous cell line. Finite cell lines are those cell lines which have a limited life span and grow through a limited number of cell generations. The cells normally divide 20 to 100 times (i.e. is 20-100 population doublings) before extinction. Cell lines transformed under in vitro conditions give rise to continuous cell lines. The continuous cell lines are transformed, immortal and tumorigenic.
- The physical environment includes the optimum pH, temperature, osmolality and gaseous environment, supporting surface and protecting the cells from chemical, physical, and mechanical stresses.
- Nutrient media is the mixture of inorganic salts and other nutrients capable of sustaining cell survival in vitro.
- Serum is essential for animal cell culture and contains growth factors which promote

cell proliferation. It is obtained as exuded liquid from blood undergoing coagulation and filtered using Millipore filters.

- Cryo preservation is storing of cells at very low temperature (-180°C to -196°C) using liquid nitrogen. DMSO is a cryopreservative molecule which prevents damage to cells.
- In order to maintain the aseptic conditions in a cell culture, a LAF hood is used. Based on the nature of cells and organism the tissue culture hoods are grouped into three types: Class I, Class II, and Class III.
- CO₂ incubators are used and designed to mimic the environmental conditions of the living cells.
- An inverted microscope is used for visualizing cell cultures in situ.
- For most animal cell cultures low speed centrifuges are needed.
- Neuronal cells constitute the nervous system. In culture the neuronal cells cannot divide and grow.
- The cells that form connective tissue (skin) is called fibroblast. The fibroblast can divide and grow in culture to some generations after which they die. All normal animal cells are mortal.
- **Organ culture**- The culture of native tissue that retains most of the in vivo histological features is regarded as organ culture.
- **Histotypic culture**- The culturing of the cells for their reaggregation to form a tissue-like structure represents histotypic culture.
- **Organotypic culture**- This culture technique involves the recombination of different cell types to form a more defined tissue or an organ.

There are certain terms that are associated with the cell lines.

These are as follows:

Split ratio - The divisor of the dilution ratio of a cell culture at subculture.

Passage number - It is the number of times that the culture has been cultured,

Generation number - It refers to the number of doublings that a cell population has undergone.

In fact these parameters help us to distinguish the cancer cells in culture from the normal cells because the cancer cells in culture, change shape (more rounded), loose contact inhibition, pile on each other due to overgrowth and uncontrolled growth.

Requirements for animal cell culture:

Among the essential requirements for animal cell culture are special incubators to maintain the levels of oxygen, carbon dioxide, temperature, humidity as present in the animal's body. The synthetic media with the sources of vitamins, amino acids and fetal calf serum are essential. Following parameters are essential for successful animal cell culture:

- **Temperature**- In most of the mammalian cell cultures, the temperature is maintained

at 37°C in the incubators as the body temperature of *Homo sapiens* is 37°C.

- **Culture media**- The culture media is prepared in such a way that it provides-
 - ✓ The optimum conditions of factors like pH, osmotic pressure, etc.
 - ✓ It should contain chemical constituents which the cells or tissues are incapable of synthesizing. Generally the media is the mixture of inorganic salts and other nutrients capable of sustaining cells in culture such as amino acids, fatty acids, sugars, ions, trace elements, vitamins, cofactors, and ions. Glucose is added as energy source- its concentration varying depending on the requirement. Phenol Red is added as a pH indicator of the medium. There are two types of media used for culture of animal cells and tissues- the natural media and the synthesized media.
 - ✓ **Natural Media** - The natural media are the natural sources of nutrient sufficient for growth and proliferation of animal cells and tissues. The Natural Media used to promote cell growth fall in three categories.
 - ✓ Coagulant, such as plasma clots. It is now commercially available in the form of liquid plasma kept in silicon ampoules or lyophilized plasma. Plasma can also be prepared in the laboratory taking out blood from male fowl and adding heparin to prevent blood coagulation.
 - ✓ Biological fluids such as serum. Serum is one of the very important components of animal cell culture which is the source of various amino acids, hormones, lipids, vitamins, polyamines, and salts containing ions such as calcium, ferrous, ferric, potassium etc. It also contains the growth factors which promotes cell proliferation, cell attachment and adhesion factors. Serum is obtained from human adult blood, placental, cord blood, horse blood, calf blood. The other forms of biological fluids used are coconut water, amniotic fluid, pleural fluid, insect haemolymph serum, culture filtrate, aqueous humour, from eyes etc.
 - ✓ **Tissue extracts for example Embryo extracts**- Extracts from tissues such as embryo, liver, spleen, leukocytes, tumour, bone marrow etc are also used for culture of animal cells.
- **Synthetic media** : Synthetic media are prepared artificially by adding several organic and inorganic nutrients, vitamins, salts, serum proteins, carbohydrates, cofactors etc. Different types of synthetic media can be prepared for a variety of cells and tissues to be cultured. Synthetic media are of two types- Serum containing media (media containing serum) and serum- free media (media without serum). Examples of some media are: minimal essential medium (MEM), RPMI 1640 medium, CMRL 1066, F12 etc.

Advantages of serum in culture medium are:

- ✓ serum binds and neutralizes toxins
- ✓ serum contains a complete set of essential growth factors, hormones, attachment and spreading factors, binding and transport proteins,

- ✓ it contains the protease inhibitors,
- ✓ it increases the buffering capacity,
- ✓ it provides trace elements.

Disadvantages of serum in culture medium are:

- ✓ it is not chemically defined and therefore its composition varies a lot,
- ✓ it is sometimes source of contamination by viruses, mycoplasma, prions etc,
- ✓ it increases the difficulties and cost of downstream processing
- ✓ it is the most expensive component of the culture medium.
- **pH**- Most media maintain the pH between 7 and 7.4. A pH below 6.8 inhibits cell growth. The optimum pH is essential to maintain the proper ion balance, optimal functioning of cellular enzymes and binding of hormones and growth factors to cell surface receptors in the cell cultures. The regulation of pH is done using a variety of buffering systems. Most media use a bicarbonate-CO₂ system as its major component.
- **Osmolality**- A change in osmolality can affect cell growth and function. Salt, Glucose and Amino acids in the growth media determine the osmolality of the medium. All commercial media are formulated in such a way that their final osmolality is around 300 mOsm.

Types of cell cultures:

Primary cell culture : The maintenance of growth of cells dissociated from the parental tissue (such as kidney, liver) using the mechanical or enzymatic methods, in culture medium using suitable glass or a plastic container is called Primary Cell Culture.

The primary cell culture could be of two types depending upon the kind of cells in culture.

- **Anchorage Dependent /Adherent cells**- Cells shown to require attachment for growth are set to be Anchorage Dependent cells. The Adherent cells are usually derived from tissues of organs such as kidney where they are immobile and embedded in connective tissue. They grow adhering to the cell culture.
- **Suspension Culture/Anchorage Independent cells** - Cells which do not require attachment for growth or do not attach to the surface of the culture vessels are anchorage independent cells/suspension cells. All suspension cultures are derived from cells of the blood system because these cells are also suspended in plasma in vitro e.g. lymphocytes.

Secondary cell cultures : When a primary culture is sub-cultured, it becomes known as secondary culture or cell line. Subculture (or passage) refers to the transfer of cells from one culture vessel to another culture vessel.

Sub culturing - Subculturing or splitting cells is required to periodically provide fresh nutrients and growing space for continuously growing cell lines. The process involves removing the growth media, washing the plate, disassociating the adhered cells, usually enzymatically.

Such cultures may be called secondary cultures.

Cell Line

A Cell Line or Cell Strain may be finite or continuous depending upon whether it has limited culture life span or it is immortal in culture. On the basis of the life span of culture, the cell lines are categorized into following types:

- **Finite cell Lines** - Like most living organisms, cells have a preprogrammed life span that determines the number of times a cell is capable of dividing. This is a natural phenomenon known as cell senescence, which makes most cell lines finite. Finite cell lines develop within primary cell cultures. The cell lines which have a limited life span and go through a limited number of cell generations (usually 20-80 population doublings) are known as Finite cell lines. These cell lines exhibit the property of contact inhibition, density limitation and anchorage dependence. The growth rate is slow and doubling time is around 24-96 hours.
- **Continuous Cell Lines** - Cell lines transformed under laboratory conditions or in vitro culture conditions give rise to continuous cell lines. The cell lines show the property of ploidy (aneuploidy or heteroploidy), absence of contact inhibition and anchorage dependence. They grow in monolayer or suspension form. The growth rate is rapid and doubling time is 12-24 hours.
- **Monolayer cultures** - When the bottom of the culture vessel is covered with a continuous layer of cells, usually one cell in thickness, they are referred to as monolayer cultures.
- **Suspension cultures** - Majority of continuous cell lines grow as monolayer. Some of the cells which are non-adhesive e.g. cells of leukemia or certain cells which can be mechanically kept in suspension, can be propagated in suspension. There are certain advantages in propagation of cells by suspension culture method.

These advantages are:

- (a) The process of propagation is much faster.,
- (b) The frequent replacement of the medium is not required.,
- (c) Suspension cultures have a short lag period,
- (d) Treatment with trypsin is not required,
- (e) A homogenous suspension of cells is obtained,
- (f) The maintenance of suspension cultures is easy and bulk production of the cells is easily achieved.
- (g) Scale-up is also very convenient.

The cell lines are known by:

- a) A code e.g. NHB for Normal Human Brain.
- b) A cell line number- This is applicable when several cell lines are derived from the same

cell culture source e.g. NHB1, NHB2.

c) Number of population doublings, the cell line has already undergone e.g. NHB2/2 means two doublings.

Characterization of cell lines:

The cell lines are characterized by their a) growth rate and b) karyotyping.

a) **Growth Rate** - A growth curve of a particular cell line is established taking into consideration the population doubling time, a lag time, and a saturation density of a particular cell line. A growth curve consists of:

- **Lag Phase** : The time the cell population takes to recover from such sub culture, attach to the culture vessel and spread.
- **Log Phase** : In this phase the cell number begins to increase exponentially.
- **Plateau Phase** : During this phase, the growth rate slows or stops due to exhaustion of growth medium or confluency.

b) **Karyotyping** - Karyotyping is important as it determines the species of origin and determine the extent of gross chromosomal changes in the line. The cell lines with abnormal karyotype are also used if they continue to perform normal function. Karyotype is affected by the growth conditions used, the way in which the cells are subcultured and whether or not the cells are frozen.

c) There are certain terms that are associated with the cell lines.

These are as follows:

- **Split ratio**- The divisor of the dilution ratio of a cell culture at subculture.
- **Passage number**- It is the number of times that the culture has been cultured.
- **Generation number**- It refers to the number of doublings that a cell population has undergone.

Table1:-Some animal cell lines and the products obtained from them

Cell line	Product
Human tumour	Angiogenic factor
Human leucocytes	Interferon
Mouse fibroblasts	Interferon
Human Kidney	Urokinase
Transformed human kidney cell line, TCL-598	Single chain urokinase-type plasminogen activator (scu-PA)
Human kidney cell (293)	Human protein (HPC)
Dog kidney	Canine distemper vaccine
Cow kidney	Foot and Mouth disease (FMD) vaccine
Chick embryo fluid	Vaccines for influenza, measles and mumps

Duck embryo fluid	Vaccines for rabies and rubella
Chinese hamster ovary (CHO) cells	1. Tissue-type plasminogen activator (t-PA) 2. β -and gamma interferons 3. Factor VIII

Applications of animal cell culture:

The animal cell cultures are used for a diverse range of research and development. These areas are:

- Production of antiviral vaccines, which requires the standardization of cell lines for the multiplication and assay of viruses.
- Cancer research, which requires the study of uncontrolled cell division in cultures.
- Cell fusion techniques.
- Genetic manipulation, which is easy to carry out in cells or organ cultures.
- Production of monoclonal antibodies requires cell lines in culture.
- Production of pharmaceutical drugs using cell lines.
- Chromosome analysis of cells derived from womb.
- Study of the effects of toxins and pollutants using cell lines.
- Use of artificial skin.
- Study the function of the nerve cells.

Somatic Cell Fusion

One of the applications of animal cell culture is the production of hybrid cells by the fusion of different cell types. These hybrid cells are used for the following purposes:

- Study of the control of gene expression and differentiation
- Study of the problem of ‘malignancy’
- Viral application,
- Gene mapping,
- Production of hybridomas for antibody production.

In 1960s, in France for the first time, the hybrid cells were successfully produced from mixed cultures of two different cell lines of mouse. Cells growing in culture are induced by some of the viruses such as ‘Sendai virus’ to fuse and form hybrids. This virus induces two different cells first to form heterokaryons. During mitosis, chromosomes of heterokaryon move towards the two poles and later on fuse to form hybrids. It is important to remove the surface carbohydrates to bring about cell fusion. Some other chemicals like polyethylene glycol also induce somatic cell fusion. Many commercial proteins have been produced by animal cell culture and there medical application is being evaluated.

CELL LINES

Cancer cells that keep dividing and growing over time, under certain conditions in a laboratory. Cancer cell lines are used in research to study the biology of cancer and to test cancer treatments.

HeLa Cells:

Henrietta Lacks and the Origin of HeLa Cells:

In 1951 a 31-year-old woman named Henrietta Lacks walked into John Hopkins Hospital in Baltimore, USA, complaining of a knot in her womb and abnormal bleeding. On the same day she was diagnosed with cervical cancer, and eight months later she was dead. This is a tragedy that has unfolded many times, but what distinguishes Henrietta Lacks's case is that cells taken from her tumour are still alive today in countless laboratories around the world. These cells have generated an entirely new branch of scientific research, and have played a vital role in the development of new treatments for many medical conditions. The story of Henrietta's ordinary life that became an extraordinary death has over the years received a number of twists, not least because while her cells have so profoundly benefited the lives of so many, her own family cannot afford medical insurance that would give them access to treatments her cells have helped to develop.

About Henrietta Lacks:

Henrietta Lacks was the great-great-granddaughter of slaves and slave holders, born to Eliza and John Pleasant on 1 August, 1920 in Roanoke, Virginia. Her name at birth was Loretta Pleasant, although none of her relatives know when or why she changed her name to Henrietta. Eliza Pleasant died in 1924 giving birth to her tenth child, after which John took the children to live with their mother's relatives. Henrietta was raised by her grandfather at Clover, Virginia and grew up helping to tend the tobacco crops her family had farmed for generations (and some continue to farm today). In 1941 Henrietta surprised many members of her family by marrying her first cousin, David Lacks. They had been raised as brother and sister on the family farm, but he was already father to her two children by the time of their wedding; Henrietta had her first child when she was 14. David moved to Baltimore looking for a better life for the family and found work at Sparrow Point shipyards. By 1943 Henrietta, their son Lawrence, and Elsie their daughter joined him and set up home at New Pittsburgh Avenue in Turners Station. Henrietta and David had three more children, David 'Sonny' Jnr, Deborah, and Zakoriyya who was born just four and a half months before Henrietta was diagnosed with cancer.

Although the move to Baltimore was intended to make life more comfortable, circumstances did not always make this so. Henrietta had five children to rise with a philandering husband who may have given her both gonorrhoea and syphilis. Elsie, who was

described by the family as ‘different’ and ‘deaf and dumb’, was placed in Crownsville State Hospital in Maryland, in 1950. She died there five years later, aged 15.

Henrietta and John Hopkins Hospital:

During Henrietta’s visit to John Hopkins Hospital on 1 February, 1951, a sample of cells from the cancer tumour was taken for research without her knowledge or consent. On her second visit around eight days later, Dr George Gey took another sample of the cells, and it is these that formed the origin of what were to become known as HeLa cells. Dr Gey, a medical researcher, had been trying for 20 years to grow human cells outside of the body to use in medical research, but his efforts were unsuccessful. Henrietta’s cells however proved to be different. They were highly aggressive and multiplied very quickly, producing a new generation of cells every 24 hours. Dr Gey had finally found the means to study cells outside of the body, thereby establishing a new branch of tissue research that offered huge potential for medical advances. Dr Gey named these cells HeLa cells, taking the first few letters from the name of their donor, Henrietta Lacks. On 8 August, Henrietta returned to the hospital in excruciating pain and remained there until her death on the segregated ward for ‘blacks’. She was buried in the family plot in an unmarked grave at Lackstown. Within a few months of Henrietta’s death, the creation of HeLa cells went into mass production at the Tuskegee Institute in Alabama, partly spurred on by the need to find a cure for the polio epidemic that had gripped America. The institute produced about three trillion HeLa cells per week and sent them to laboratories around the world.

HeLa Cells in Medical Research:

Shortly after Henrietta’s death, Dr Gey along with a fellow researcher in Minnesota discovered that HeLa cells are susceptible to polio. In February 1952 Jonas Salk had developed a vaccine for the disease but needed human cells to test and ensure its safety. HeLa cells were a perfect candidate and were able to play a significant part in the development of the vaccine. Since then HeLa cells have been used in numerous medical research programmes and have proved to be an untold benefit to humanity. HeLa cells have made a contribution in the following areas:

- Due to a laboratory accident involving HeLa cells, in 1953 scientists in Texas were able to see human chromosomes for the first time. Using HeLa and mouse cells, two British scientists created the first human-animal hybrid cells in 1965.
- Cell culture techniques based on those developed using HeLa cells led to the birth of Louise Brown in 1978, the first ‘test tube’ baby.
- In the 1980s HeLa cells were infected with HIV by Richard Axel, which led to major advances in the understanding of the virus.
- Perhaps the most poignant area of research HeLa cells were used in was the discovery of a vaccine for what was the cause of Henrietta’s death. In the 1980s the German virologist Harold zur Hausen discovered Human Papilloma Virus HPV-18, one of the main

causes of cervical cancer, and found that Henrietta had been infected with a particularly virulent strain. Through his work with HeLa cells and others, Hausen won the Nobel Prize for the cervical cancer vaccine he developed.

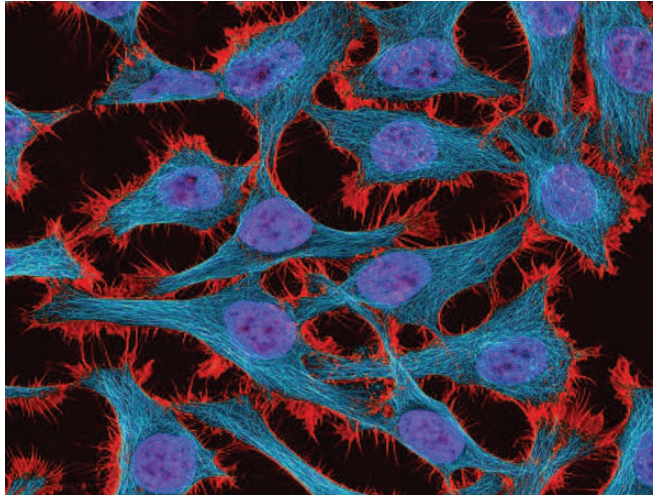


Fig: Multiphoton fluorescence image of HeLa cells stained with the actin binding toxin phalloidin (red), microtubules (cyan) and cell nuclei (blue).

These areas of medical research represent just a fraction of those that HeLa cells have been part of. Since Henrietta's death there have been more than 50 million tonnes of HeLa cells produced and their use has been acknowledged in more than 60,000 scientific papers, with 10 new ones added each day. HeLa cells are so integral to medical research there is now a thriving billion-dollar medical industry based around them, with over 11,000 patents logged with the US Patent and Trademark Office as a result of their use. With a vial of HeLa cells costing around £ 175, many people have become very rich, and a lot of careers have been launched due to their invaluable role in medical research.

Henrietta's Family:

The story of HeLa cells is one of both scientific and financial success, but for Henrietta's remaining family the story has been very different. Henrietta left behind a young family with very limited financial means and without access to the advantages of education. In 1955 they also lost their sister Elsie who died at the age of 15 at the Crownsville Hospital. They later learned she had been abused there and may have had holes drilled in her head as part of experiments. The Lacks family story is one of many hardships, but for scientists it was not a story that had a direct relevance to their work with HeLa cells. Because of this, and partly to protect the anonymity of the source of HeLa cells, there was a fundamental absence of communication with family members. For 20 years the family had no idea their mother's cells were still alive in laboratories throughout the USA and beyond. The first time the Lacks family became aware of the existence of HeLa cells was in 1973, when someone who recognized the surname asked if they were related to Henrietta, the cells' donor. Around the same time,

medical researchers asked the Lacks family for blood samples, which they believed were to test for signs of cancer. However, they were not told the samples were to help study the genetic structure of HeLa cells. After obtaining the blood samples the researchers did not contact the family again, so they were left to wonder if they were at risk of suffering the same fate as their mother. Apart from the lack of communication, at the heart of the way Henrietta's family has been treated is the issue of money. While HeLa cells have brought many medical advances and wealth amongst the medical establishment, none of that wealth has gone to Henrietta's family. In fact the Lacks family cannot afford healthcare, so cannot buy the medicines their mother's cells have helped to develop. As well as financial considerations, there is the contrast between the plight of the Lacks family and the career success of those who worked with HeLa cells, as well as those whose lives have been improved and extended by medical treatments brought about by their use.

Commemorating Henrietta Lacks:

The name of Henrietta Lacks is still relatively unknown with little being done to recognize her posthumous contributions to medicine. However, some encouraging steps have been taken. In 1996 the Morehouse School of Medicine in Atlanta, Georgia and the mayor of Atlanta formally recognized Henrietta and her family for their role in the development of HeLa cells. For a while following this, Henrietta's home district of Turners Station commemorated her life on 1 February each year. Soon afterwards the mayor of Baltimore declared 11 October Henrietta Lacks Day. In 1997 the American Congress passed a resolution (sponsored by Rep Robert Ehrlich, whose second district includes Turner Station), acknowledging Henrietta and HeLa cells' contribution in medical advances. After 59 years Henrietta's unmarked grave finally received a headstone. On 29 May, 2010 Dr Roland Pattillo of Morehouse School of Medicine donated two headstones, one for Henrietta and one for her daughter, Elsie. The inscription on Henrietta's headstone reads:

Henrietta Lacks

August 01, 1920 - October 04, 1951

In loving memory of a phenomenal woman,
wife and mother who touched the lives of many.
Here lies Henrietta Lacks (HeLa). Her immortal
cells will continue to help mankind forever.

Eternal Love and Admiration, From Your Family

Perhaps the most promising acknowledgment for the Lacks family is the publication of a book, 'The Immortal Life of Henrietta Lacks' by Rebecca Skloot, which follows the many strands that form the complex story of Henrietta's life, HeLa cells, and the aftermath of her death for the remaining family members. Ms Skloot has also set up the Henrietta Lacks Foundation so that some of the proceeds from her book can help provide the Lacks family with medical insurance, as well as helping other African Americans who wish to follow a

career in science or medicine.

Although there is evidently a discrepancy between the success that HeLa cells have brought for the medical community and the plight of the Lacks family, this is not to suggest they were treated any differently than anyone else in their circumstances. In the 1950s both social and medical ethics were different than they are now, and it was standard practice to take tissue samples for research without the patient's consent. The plight of the Lacks family may also create the notion of an uncaring medical establishment exploiting HeLa cells with a disregard for the Lacks. But this would be to overlook the efforts medical researchers have undertaken in order to develop better treatments and medicines for innumerable conditions while working with HeLa cells.

However, the story of Henrietta Lacks and her family still lies uncomfortably because it raises many unresolved issues, such as what rights patients have over their own cells, and what responsibilities the medical profession has towards patients if their cells lead to medical breakthroughs and profits. In the case of Henrietta, her daughter Deborah Lacks summed up the issue succinctly when she said: Truth be told, I cannot get mad at science, because it helps people live, and I'd be a mess without it. But I won't lie. I would like some health insurance so I don't got to pay all that money every month for drugs my mother's cells probably helped make.

Henrietta Lacks: the mother of modern medicine

Henrietta Lacks, a 31-year-old mother of five, died of cervical cancer on 4 October 1951; and while her disease was a tragedy for her family, for the world of medical research – and beyond that, every one of us on the planet – it was something of a miracle.

Because, in the years since her death, Lacks's cells – taken from her tumour while she was undergoing surgery – have been responsible for some of the most important medical advances of all time. The polio vaccine, chemotherapy, cloning, gene mapping and IVF: all these health milestones, and many more, owe everything to the life, and death, of a young mother.

Lacks's cells – known as HeLa, using the first two letters of each of her names – became the first immortal human cell line in history. Scientists at the hospital where she died, Johns Hopkins in Baltimore, had been working for years to try to start a continuously reproducing cell line – but the cells always died. Lacks's were the first that “took”, introducing a constantly reproducing line of cells that are literally, to give them their scientific definition, immortal. (Ordinary cells taken from a human body and kept in a lab have a limited life span; however, an immortal cell line is cultured in a particular way so it has the ability to proliferate indefinitely.) Quite why hers were the cells that survived and reproduced, when those of hundreds of other patients had died, is unclear – but the best guess is that the reason was linked to the ferocity of her tumour, which seems to have been made more virulent by the fact that she also suffered from syphilis.

As soon as it was clear that HeLa would continue to reproduce, all kinds of research and experiments suddenly became possible. For a start, having living cells available outside the human body meant doctors could watch cell division taking place, and could also see how viruses behaved inside the cells. What's more, it was possible to expose the cells to conditions that wouldn't have been ethical if they were inside a human body – for example, doctors could bombard them with carcinogens, and watch the results.

In the years since 1951, HeLa cells have been exposed to endless toxins and infections; they've been zapped by radiation, and tested with countless drugs. And all this – and much, much more – has led to hundreds, if not thousands, of new pieces of knowledge, and helped to shape the way medicine moved in the second half of the 20th century and the first decade of this one. And there are certainly plenty of HeLa cells to go round, these days: one researcher has estimated that if you laid them all end-to-end, they'd wrap around the planet at least three times. No one would be more surprised to know this than Lacks (who, in her lifetime, stood about five feet tall). But for decades, while HeLa cells were routinely being used in laboratories around the world, and were being hailed as pivotal in breakthrough after breakthrough, no one seems to have stopped to think about the person behind them. Then, 37 years after Lacks's death, a 16-year-old schoolgirl called Rebecca Skloot was sitting in a biology lesson when her teacher explained how cancer begins, and said the process had been learned from studying cells in culture – HeLa cells. The cells, said the teacher, came from a woman called Henrietta Lacks.

When the class was over, the other students filed out – but Skloot hung around. “I said to my teacher: who was this woman Henrietta Lacks? Where was she from? Did she have any kids? But all the teacher knew was that she was black, and that she had died in 1951 from cervical cancer.” After school, and a degree in biological science, Skloot, who is promoting her book about what she calls the “immortality” of Lacks, devoted herself to finding out the truth behind the HeLa cells – and what she uncovered was a tale that is immensely moving. It's also a story that has captured the public imagination: since its publication in February in the US, Skloot's book has never been off the New York Times bestseller list. What Skloot found out puts the American healthcare system, and beyond it scientists everywhere who depend on patient goodwill, but fail to communicate effectively, in the dock. Because what she found out was that, while Lacks's cells were changing the face of modern medicine, her husband and children not only knew nothing about it – they were also without adequate healthcare themselves. “What most people are most shocked at is that Henrietta's cells were taken without her knowledge, and without her consent,” says Skloot. “But that's standard practice, here in the UK as in the US. If you sign a general consent form before surgery, any sample cells removed may be used for research later, and the doctors don't have to let you know. “The general standpoint of medical science is that cells taken from an individual and used for research benefit the common good, so it's OK to use them. But the Lacks story shows that isn't true – certainly not in America, anyway. Because Henrietta's cells were used

to develop medical treatments – but those treatments were only available to people who could afford medical insurance, and impoverished families like the Lackses were exactly the sort of family who couldn't." To make matters worse, Lacks's cells were making some people – pharmaceutical companies – rich. More specifically, cell banks and biotech companies were retailing vials of her cells – the current going rate for a tube of HeLas is around US \$260 (£174). But not a penny of the profits her cells had helped to generate went to her descendants: and while their mother's cells were soaring to worldwide scientific acclaim, fortunes in the Lacks family had plummeted. It wasn't, of course, all that surprising. The Lacks family was not, and is not, wealthy. Henrietta's husband Day (with whom she'd had her first baby aged 14) worked in a steel mill in Baltimore, making about 80 cents an hour. Life in their household was tough enough, with five children to feed, even before their mother got cancer. What chance, really, would they have with her gone? Skloot's testimony – and she has interviewed hundreds of people for her book – reveals a tragic tale. Lacks clearly knew how precarious her kids' lives were going to be once she wasn't around any more. As she was dying, the doctors told her husband that she was too ill to have visits from her children – the youngest of whom was just 13 months old. So instead, Day Lacks would take them to play in a garden across the road from her ward. And despite being in excruciating pain, Lacks would drag herself out of bed to the window, and press her face against the glass looking at the children she knew she'd never hold in her arms again.

Her last request to Day, Skloot discovered, was to ask him to "take care of them kids . . . don't let nuthin happen to them". And Day tried his best, but the odds were stacked against him. Their eldest daughter Elsie, who had developmental problems, was already in the Hospital for the Negro Insane, and died there soon after her mother. A son, Joe, dropped out of school and later stabbed another boy, and was sentenced to 15 years in jail. Daughter Deborah was a teenage mother who later left her husband after he beat her up. By the time Skloot caught up with them all in 2000, nearly everyone was in poor health: Day had prostate cancer and asbestos-filled lungs; one son, Sonny, had a bad heart; Deborah had arthritis, osteoporosis, nerve deafness, anxiety and depression. None had medical insurance cover and money for treatment. "If our mother is so important to science," another son, Lawrence, asked Skloot, "why can't we get health insurance?"

It was Lawrence's wife, Bobette, who was the first member of the Lacks family to hear about Henrietta's cells. By chance, she had met a cancer researcher – and when she told him her name, the researcher remarked that he was working in the lab on some cells that came from a woman named Henrietta Lacks. Bobette said that had been her mother-in-law's name – but it couldn't be her because she'd been dead for years. And then the researcher explained that the cells had been growing for years – ever since 1951, in fact. It was a crass way for her family to find out what had happened to Lack's cells, but in fact they would have found out anyway. The cell line had been contaminated, and scientists realised they needed to test Henrietta's descendants to work out what had contaminated them. "But when they came back

to take cell samples, they didn't explain properly to the Lackses what was going on," says Skloot. "Henrietta's children thought they were being tested for the cancer that had killed her . . . Deborah waited for months thinking she was going to find out whether she'd die the same agonising way her mother had."

One of the biggest issues her book raises, says Skloot, is how important it is for doctors and other health workers to communicate effectively with patients and their families. "If you spoke another language and you needed to see the doctor, you'd be provided with a translator – but if it's the science you don't understand, there's no one there to translate for you, so you go away simply not knowing what's been said. I think there should be science translators, who are trained to communicate complicated medical stuff in a straightforward, easily digestible manner. It would have made a huge difference to Henrietta's family." In many ways, Skloot became the "explainer" the Lacks children so desperately needed. "Usually when you're working as a journalist you're asking people about their story – but when I met the Lacks family, I was telling them about theirs," says Skloot. Another question her book raises – and it's likely to be even more pertinent in the future, as medical research becomes a bigger and bigger, multi-billion-pound industry – is how much right do we have over the raw materials of our physiology? What rights do the providers of the original sample – or their families – have if their cell lines are later found to be worth patenting?

But the biggest point Skloot wants to make is that behind every test-tube of cells there lies a real, human story. "Tissue is so often dehumanised – it's referred to in medical reports and documents, and no one ever seems to remember that for every single biological sample that's used in any laboratory, anywhere, there's a person." Perhaps surprisingly, she says the people who most conspire to make this the way it is – the very scientists whose experiments require human cells and tissue – have greeted her book favourably. "One researcher said he'd never thought about the person behind the cells and now he knows the story, when he's working on HeLa cells he feels there's a ghost in the lab – the ghost of Henrietta." Meanwhile in a cemetery in Virginia, where Henrietta Lacks was buried in an unmarked grave, a memorial has at last been erected. It is dedicated to the memory of a woman who, it says "touched the lives of many"; and no truer a sentence has ever been inscribed.

SARCOPTES SCABIEI (MITES)

The source of the non conventional drug Psorinum is the alcoholic extract of the Scabies scrub, Slough and Pus cells. In course of our research work ample evidence is found for existence of some anticancer agent in this drug causing shrinkage of tumor and other favorable indication in extending the life span of the patient. So it becomes imperative to encompass details about scabies and mites which have immense possibility of being anti-cancer agents.

The discovery of the itch mite in 1687 marked scabies as the first disease of humans with a known cause. The Italian biologist Diacinto Cestoni showed in the 18th century that scabies is caused by a tiny mite (like a tiny insect) called *Sarcoptes scabiei*. The disease produces intense, itchy skin rashes when the impregnated female tunnels into the stratum corneum of the skin and deposits eggs in the burrow. *Sarcoptes scabiei* is a skin parasite causing severe itching and infections. It belongs to the kingdom animalia, class of arachnida, subclass acari and family Sarcoptidae. It gets its name from the Latin word “scabere” which means to “scratch”. *Sarcoptes scabiei* is a parasitic arthropod that lives within the subcutaneous tissues of skin on humans, causing the condition known as scabies; similar mites cause what is called “mange” in wild and domestic animals. This mite is distributed worldwide, and can affect all socioeconomic groups. Scabies mites are generally host specific and *S. scabiei* is dependent on humans for its life cycle with no benefit to the host. There are at least 300 million cases every year worldwide. All ages of men and women especially in crowded and unhygienic conditions are at risk. Humans as well as other mammals such as wild and domesticated dogs and cats as well as ungulates, wild boars, bovids, wombats, koalas, and great apes are affected.

History of Discovery : Scabies is an ancient disease based on archeological evidence from Egypt and the Middle East, scabies is estimated to date back over 2,500 years. The first recorded reference to scabies is believed to be from the Bible in 1200 BCE. Later, the Roman philosopher Aristotle reported on “lice” that would “escape from little pimples if they are pricked” in the fourth century BCE (Roncalli). The Roman physician Celsus credited with designating the term “scabies” to the disease and describing its characteristic features (Roncalli). The parasitic etiology of scabies was later documented by the Italian physician Giovanni Cosimo Bonomo (1663-1969 ADE) in his famous 1687 letter, “Observations concerning the fleshworms of the human body” (Roncalli). With this (disputed) discovering, scabies became one of the first diseases with a known cause.

Mode of Transmission : The majority of scabies cases are transmitted by skin-to-skin contact with persons carrying the scabies mite. Less often, scabies can be transmitted by sharing of clothes and bedding. Theoretically, touching an object that a mite is on is a third mode of transmission; however, this is not at all common.

Host Immune Response : Scabies mites when burrow into the human skin, the eggs,

mites and feces trigger a host immune response. Like an allergic reaction, this autonomic immune response results in a rash, itching, and occasionally a fever. However, this immune response does not occur until days to weeks after infection, due in large part to the fact that scabies mites are genetically encoded with immunological ‘weapons’ that prevent the host from responding to its presence (Burkhart). Mites cannot survive longer than 3 days without a human host. Once infected, the scabies lifecycle will continue until medication is used to treat the disease. The associated skin disease characteristic of scabies develops due to delayed immune system hypersensitivity. The incubation period for this itching and rash is usually 2-6 weeks. However, in individuals with prior exposure to scabies, the incubation period is much shorter: as little as 1 to 4 days.

Morphology:

Mites have a cream-colored body, bristles and spines on their back, and four pairs of legs. Their bodies are covered with fine lines and several long hairs. The mites have no eyes, and they have short and thick legs, with the first two pair of legs stalked. The immature stages of the scabies mite are comprised of a six legged larval stage, followed by 2 nymphal stages that have eight legs, and each stage resembles the adult mite. The female mite is bigger about 0.33-0.6 mm long and 0.25-0.4 mm wide compared with the male 0.2-0.24 mm long, 0.15-0.2 mm wide. 8 short legs, posterior 2 pairs do not extend beyond the body margin; unsegmented pedicels; mouthparts have a rounded appearance. The female mite has scattered on the dorsal surface some short blunt spines, which aid her in maintaining her position within the tunnel. Scabies mites burrow into the outer layers of human skin, causing itching, a rash, and sometimes skin sores. The scabies mites are spread through close contact with an infected person, such as by touching or by sleeping in the same bed. The female mites tunnel into the skin and lay eggs. About 40-50 eggs are laid in the lifetime of a mite. The eggs hatch into larvae after 3-4 days; these then grow into adults within 10-15 days. Less than one in 10 eggs becomes an adult scabies mite. Most of the symptoms of scabies infestation are due to the immune system response to the mites themselves, their saliva, their eggs or their faeces.



Figure 1 and 2: Sarcoptes Scabiei (Scabies mites)



Figure: 2



Figure 3: Infection caused by *Sarcoptes Scabiei* (Scabies mites)



Figure 4: Difference between Scabies, Acne and Mosquito bites infections.

Life cycle

The entire life cycle of the mite occurs over 10-17 days. Newly mated females take approximately an hour to burrow into the outer layer of human skin and excavates a tunnel. *Sarcoptes scabiei* goes through four stages in his life cycle such as egg, larva, nymph and adult. The life cycle starts, when an adult female mite gets in contact with our skin. It crawls to crevices such as elbows, feet, fingers and genital area. It penetrates the skin and burrows a tunnel. It can slice skin with its sharp front legs and mouthparts. The other legs it uses for holding on to the skin with suckers on each leg. It takes about 30 minutes for it to burrow into the skin, eating the skin and tissue fluids that ooze from their excavations. Each tunnel contains only one female, her eggs and faeces. It then continues to drill horizontally across the skin laying eggs along the way. The tunnels are usually shaped in a zigzag on the skin surface. It lays 2-3 eggs per day for two months after that it dies. Eggs are oval in shape and 0.10 to

0.15 mm in length. After 48 hours the eggs hatch and the larval stages dig their way to the surface of the skin, where they immediately burrow. This burrow may only be a short distance into the skin, and they find hair follicles where they feed and molt into eight-legged nymphs. In order for the nymph to become an adult male it molts once which takes about ten days. To become a female it has to molt twice which takes about 17 days. Since the nymph has more time to eat and grow the females are larger than males. Larval and nymphal stages remain in these moulting pockets feeding on fluids secreted from the follicles before moulting to the adult stage. Newly moulted male and female mites construct short burrows <1mm before mating. Mature female and male mate only once. The sperm keep the female fertile for the whole two months that it lays eggs. Males do not usually burrow into the skin but only crawl and feed on it.

After fertilization, female mites wander on the skin to seek a suitable site for a permanent burrow; the transfer of a female to another host at this stage will initiate a new infection. A fertilized female mite can only initiate successful scabies infections. Female mites rarely leave their burrows, and if removed by scratching and remain undamaged, they will attempt to burrow again. During an infection the number of mites increases rapidly, and then drops off, leaving infected persons with a relatively stable mite population of 15-20 females. The mortality rate of mites is high, 90% of mites that hatch will die, and mites removed from their host can only live a short time.

The movement of *Sarcoptes scabiei* and eggs inside the tunnels cause local inflammation. This allergic reaction causes very intense rashes. People who have never been exposed to scabies develop allergic response within six weeks. Those who have had scabies previously will get the rash within a few days. On average there are only a few fertile female mites per infected person. These kinds of infections can be harmless and the victim might not even notice it. Scabies spreads easily through skin-to-skin contact. Mites can also crawl long distances. If we scratch an infected area, they get inside our fingernails. Then if we touch common objects like laptops, the mites can drop there and infect others.

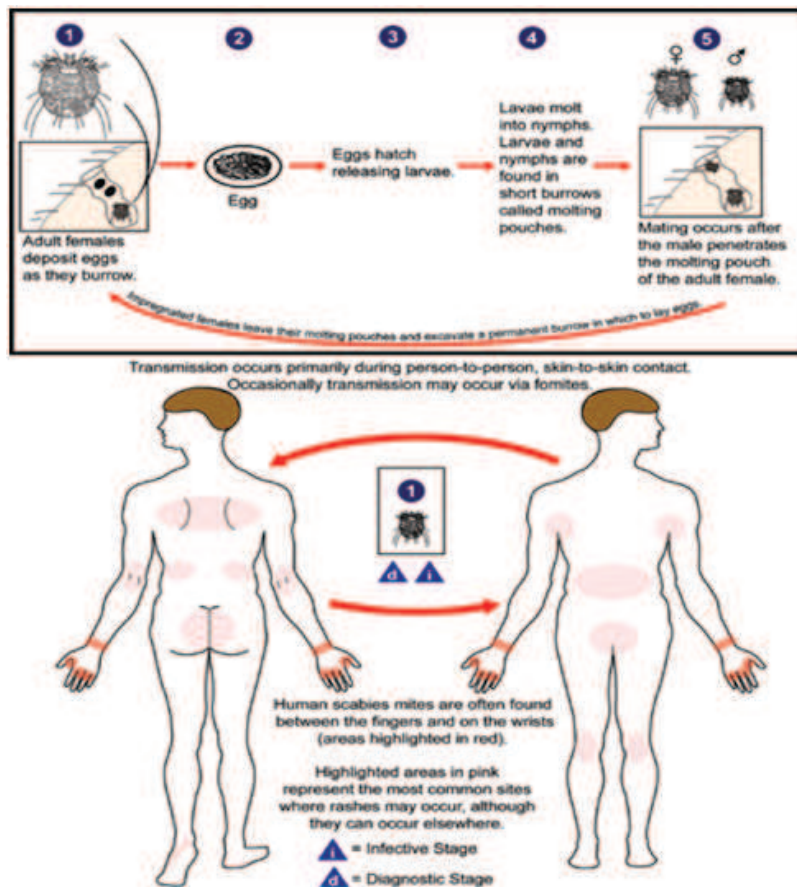


FIG 1: Life cycle of *Sarcoptes Scabiei*

Epidemiology

- Approximately 300 million cases of scabies occur worldwide each year
- Incidence is increased during wars, famine, and social upheaval
- Scabies is most prevalent among young children, institutionalized patients, impoverished persons, displaced populations, refugees, migrant workers and persons living in crowded conditions

Causes and risk factors

Causes:

- Infestation with the human mite *S. scabiei*

Risk factors:

- Close or direct skin-to-skin contact, including sexual contact, with an affected person
- Overcrowded conditions
- Immigration

- Poor nutrition
- Poor hygiene
- Homelessness
- Dementia
- Crusted (Norwegian) scabies is associated with impaired cellular immunity and is seen in patients with human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), human T-lymphotropic virus infection, lymphoma, or leprosy and organ transplant recipients; patients with Down syndrome also may be affected

Associated disorders

In the developing world, chronic scabies infestation is associated with secondary streptococcal infection and glomerulonephritis.

Pathogenesis/clinical signs

- Initial lesions occur on the hand, elbows, axillary or inguinal regions
- Lesions are initially erythematous, then become papular; papules rupture and skin becomes crusty
- Alopecia, thickening of the skin, pruritus
- Secondary bacterial infection, allergic reaction, self-mutilation may occur

Transmission

Transmission occurs primarily by the transfer of the impregnated females during person-to-person, skin-to-skin contact. Occasionally transmission may occur via fomites (e.g., bedding or clothing). Human scabies mites often are found between the fingers and on the wrists.

Diagnosis

Clinical Presentation

Scabies infestations can present different clinical pictures and may be difficult to diagnose. The initial infestation may remain undetected for a month or more, before sensitization develops and an immunological response in the host is triggered. The allergic reaction is from components of the mite's faeces, skin moults, saliva or moulting fluids diffusing into the tissues of the host from the burrows. The patient can experience severe itching all over the body, and especially at night. Large areas of the body can be covered by a rash that can last for weeks but which will not (or only rarely) coincide with the areas of mite infestation. Eruption of the skin into small itchy lesions may occur in conjunction with the rash. Scabies mites tend to burrow into the skin where there is a natural crease and the host's reaction will be minimal. The hands, and webbing between the fingers, the wrists, and elbows are common areas. From the surface of the skin, the tunnels appear as grayish pencil marks; in darker skin the tunnels appear paler. The severe itching and scratching can lead to

secondary infections and, in cases of heavy infestations anemia can develop. There has been no transmission of disease pathogens associated with this mite.

Untreated scabies infestations, especially in infants, immobilized geriatric patients, AIDS and other immunologically compromised patients can support huge numbers of female mites. The patient's skin may become crusted on the surface, with the underlying layers soft and honeycombed with tunnels, these infections are referred to as "Norwegian" or "crusted scabies". Patients with this advanced state of infection can act as a source for local epidemics in health care facilities. In some cases, scabies infections in nursing staff or family that have had contact with the patient will lead to diagnosis of the primary patient. Reinfected patients will develop an immediate itch when another scabies infection is initiated.

Laboratory Diagnosis

Diagnosis is done by finding mites or their tunnels. Skin scrapings are examined with a compound light microscope for the presence of mites, eggs or faeces. A glass slide mount is prepared, using dilute potassium hydroxide or lactic acid to mix with the skin scraping. This aids in clearing any thick layers of skin cells in the sample to reveal any evidence of the scabies mite, but clearing may take some time (hours to days). If no mites are seen but lesions strongly suggest sarcoptic mange, response to treatment may be used to reach a clinical diagnosis.

Treatment & Control

Drug of choice for scabies

Permethrin-

- Used because of its relative safety and low irritant
- Is safe for use on the head and neck of children less than two years old

Once diagnosed, most scabies infections are easy to control. In most cases, itching may persist for a week or more after the treatment, but this is not necessarily a sign of treatment failure. Reexamination of the patient at four weeks after the treatment is appropriate. At the commencement of the treatment, bed linen and underwear of the patient should be washed in hot water and hot tumble dried, but there is no need to treat furniture or rooms with an insecticide. A common problem of treatment failure is insufficient coverage of the body with the scabicide, and resistance is rare. For patients diagnosed with crusted scabies, the patient should be isolated, and barrier nursing implemented throughout the treatment. All individuals that have had significant contact with the primary patient should also be treated. Scabies is highly contagious in overcrowded situations and close contact with infected individuals should be avoided. Touching, shaking hands, or sharing beds and contaminated objects of an infected person are common modes of transmission.

People who already have other diseases and thus weakened immune systems can develop crusted scabies (also known as Norwegian scabies). It is characterized by thousands of mites causing severe itching and rash. Norwegian scabies is treated with oral dose of Ivermectin

which has some severe unwanted side effects.

Public Health Considerations

- People can develop a self-limiting infestation with *S. scabiei* from dogs. The lesions that are produced will be highly pruritic but usually clear without the need for specific treatment for the mite infestation. If lesions persist or are particularly uncomfortable, a dermatologist should be consulted.
- People also develop infestations with *Sarcoptes scabiei* var. *hominis* following contact with other infested people. Dogs are not always the source of human scabies, particularly when institutional outbreaks occur.
- Human scabies is considered a sexually transmitted disease although any close contact between individuals may facilitate transfer of mites and establishment of a new infestation.

DRUGS – YESTERDAY-TODAY & TOMORROW

Existence of life in this planet would have been impossible without remedial substances which may be of plant origin, metallic origin or organic origin. From times immemorial plants, mineral product, organic substance provided mankind many measures to relieve them from distress.

India the most ancient civilization has its own rich heritage of culture, education which was inherited through last 5 thousand years. The country in the lower meridian has sufficient sunlight, rains, water, fertile soil, mountain greeneries, walk of different cultures landed on its path. Aryans invaded India with their culture and push Dravids downwards with their culture and in this type of admixture, India had abundance in all kinds of medicine product, be it of plant origin, animal origin or mineral origin. More than 5000 plants out of about 15000 species found in India are believed to have medicinal properties of some description or other and have been enumerated in the literature of indigenous medium e.g, Rigveda was written as early as 4500 BC. Ayurveda which is thought to be written in 2500 BC. These ancient treasures have definite properties of medicinal plants and their uses in details.

Use of indigenous medicinal plant in Indian medicine flourish till the invasion of India by the British still then many western scholar regarded the India system of medicine as a rich mine of knowledge from which many useful things could possibly been un-earthed.

Ayurveda (“the complete knowledge for healthy long life”) or ayurvedic medicine is a system of traditional medicine and a form of alternative medicine, native to India (Chopra, 2000). In Sanskrit words ayus, meaning “longevity”, and Veda, meaning “knowledge” or “science”. Following the system would help to ensure a long life, which is considered to be the instrument for achieving righteousness (dharma), wealth (artha) and happiness (sukha). The Ayurveda remains one of the most ancient and yet living traditions practiced widely in India, Ceylon and other countries have a sound philosophical and experiential basis (Chopra and Doiphode, 2002). Atharvaveda (around 1200 BC), Charak Samhita and Sushrut Samhita (1000–500 BC) are the main classics that give detailed descriptions of over 700 herbs. Indian healthcare consists of medical pluralism and ayurveda still remains dominant compared to modern medicine, particularly for treatment of a variety of chronic disease conditions (Morrison, 1988).

Ayurveda is similar to Galenical Medicine in that it is based on bodily humours (dosas) and the inner life force (prana) that is believed to maintain digestion and mental activity. The living and the non-living environment, including humans are composed of the elements earth (prithvi), water (jal), fire (tejac), air (vayu) and space (akasa) that compose the universe, including the human body. Chyle or plasma (called rasa dhatu), blood (rakta dhatu), flesh (mamsa dhatu), fat (medha dhatu), bone (asthi dhatu), marrow (majja dhatu), and semen or female reproductive tissue (sukra dhatu) are held to be the seven primary constituent of the

body (saptadhatu). Ayurvedic literature deals elaborately with measures of healthful living during the entire span of life and its various phases. Ayurveda stresses a balance of three elemental energies or humors: Vayu vata (air and space- “wind”), pitta (fire and water- “bile”) and kapha (water and earth – “phlegm”). According to ayurvedic medical theory, these three substances - are important for health because when they exist in equal quantities, the body will be healthy and when they are not in equal amounts, the body will be unhealthy in various ways. One ayurvedic theory asserts that each human possesses a unique combination of doses that define that person’s temperament and characteristics. In ayurveda, unlike the Sankhya philosophical system, there are 20 fundamental qualities inherent in all substances. Surgery and surgical instruments were employed from a very early period, Ayurvedic theory asserts that building a healthy metabolic system, attaining good digestion and proper excretion leads to vitality. Ayurveda also focuses on exercise, yoga and meditation for an understanding of these traditions, the concept of impurity and cleansing is also essential. Illness is the consequence of imbalance between the various elements and it is the goal of the treatment to restore his balance.

Study of Indian indigenous drugs first began in the early part of the last century Sir William Jones wrote a memoir entitled botanical observation of selected plants in 1868. the first pharmacopoeia of India was published under the guidance of ‘warring’ which mainly described the medicinal usage of indigenous plants. Nearly 3/4th of the drug mentioned in British pharmacopoeia grows here in a state of nature and other can be grown occasionally. Moreover this country is of older civilization which enriches people with medicinal application in the form of diet. Drug substance grows here in a state of nature which is very smooth and easy. A shower of test from different corners of the country flew through years and inspite of the illustrations work by Kirtika and Bash. Sir R N Chopra and various other Indian authors have admitted that systemic pharmacological studies with most of the indigenous medicinal plants still remains as an unexplored mine and it may be said that inspite of global studies on pharmacological action of indigenous plants, organic matters and metals may posses a medicinal value without being of any practical use.

With the advancement of chemistry and other allied branches of science, the proper valuation of the traditional medicine is yet far off. Even scientists are very much sceptic about the possibility of any valuable contributions of traditional medicines to the development of therapeutics. But it must not be forgotten than wonder drugs have been shown to work here in this institute for traditional cure. Therefore the probability of finding newer useful agents from plants, metals, organic matter sources cannot be denied.

Keeping this in mind thorough research had been taken to find such elements (organic spasm), extract of mite- antigen protein and chemical. Pharmacological studies have not been carried out but good clinical reports have been found in terminal care patients (suffering from malignancy). It had been found in the history that Indian sage Charaka Sushruta made some exemplary contribution to evolutionary medicine.

Similarly K K Chen (1974) a Chinese sage contributed towards development of medicine. It stands to reason that all these medicine ingredients cannot possess the wonderful virtues attributed to them. But it is believed that there are some of these which might rightly deserve the reputation they have earned as cures. In order to determine what these were and what medicinal properties they possess the study of these plants began in the early part of the last century.

The early study mainly conferred to collection of available data from the literature of indigenous medicine as well as getting information about their popular use as house hold remedies by the people. Various surveys have been done in different parts of the country with their chemical analysis. But number is too meagre to carry out work on all medicinal products.

Experimental work on pharmacological side to determine the action of these active principles needs modern laboratories with equipment but which is not possible at the stage with moderate effect. But if the resources could be developed and utilized and finished product manufactured, treatment of any diseases could be brought within the means of the Indian patient's whose economic condition is unfortunately of very low order.

A number of important medicines (traditional) having medicinal properties, recently discovered in the nature, were so far been not cultured in India. Cultivation of such drugs is very important for an economic point of view and scientific research in the direction could be very fruitful.

But the technique used in pharmacology, microbiology, immunology, molecular biology, genetic and allied science, physiology are not much developed for utilization of these drugs.

It is a matter of respect though our country is pregnant with ideas but particularly no interaction is shown in the line of treatment with traditional drug remedies used by Indigenous medicine.

The objective has been to discover remedies from the claims of Ayurveda, Tibbi and other indigenous resources, suitable for employment by exponent of western medicines. But the knowledge of many of the effective remedies was lost while a number of uncertainties crept in. Belief in their efficacy originates from drug history of mankind. In some cases, treatment of the population is based on chemical data. Some of these drugs have been shown to be of great practical utility and have actually brought into use. They have medicinal properties which are to be brought as cheaper substitute for pharmacological and therapeutic use.

When it was felt that our material has anti-cancer activity then we have divided it in two parts and carried out the study. One part is used for human trial development and the other part is used for drug development. In the meanwhile we started studying the steps associated to develop a new drug. A book on how Penicillin was discovered emboldened our activity, knowledge and strength towards developing anticancer drug. We however deeply felt that the material which we are administering on our patients need to be established pharmacologically by pharmacodynamics and pharmacokinetic study within a short period of time.

Drug discovery & development is a costly process which a ordinary person cannot afford without support without being big MNC giants. Many compounds that are screened initially fail to make it to the next stage of development. So it is a risky task to perform. Generally to develop a drug it requires approximately 12- 15 years before it is ultimately launched in the market by Pharmaceutical companies and requires huge investment. Drug development involves six and half years, preclinical testing, toxicity study, one and half years of phase one, to see safety in healthy volunteers, two years of phase 2, with few hundred patients, phase 3, with 1000's of patients and one and half year for drug authority review. After approval, the drug undergoes phase 4 testing and more safety and efficacy data is collected.

Most of the drug development process i.e. drug discovery was done in the western countries In India the clinical trial phases were mostly run before the stickiness of the patent law. Most of the patented compounds were manufactured by doing small researches like BA/ BE testing, biosimilars etc. and apply for ANDA applications to drug authority. But after the stickiness of patent law Pharmaceutical companies have to find new novel leads and drug candidates.

Basically I did not belong to allopathic stream. But there was an urge of scientific attitude inside me and a thrust to do scientific research on cancer for the humanity for which I have to struggle hard to find out the root cause of the disease.

Where in one side drug development was in its extreme stage with most sophisticated instruments and facilities, we have to do our works with ancient knowledge as we lack proper scientific facilities and enough funds in our country. But our activities were being highly appreciated by American society of clinical oncology. So in the context of a practical field work, a country, which does not possess adequate arms, ammunitions and trained soldiers sometimes, has to take resort to the strategy of guerrilla warfare. Similarly, we have to take up guerrilla warfare instead of recognised conventional warfare with the ancient know how. We hope in the near future, the scientific world will study and do extensive work on this topic.

As we studied with ancient methodology and stone stool technology, at that time there was no scientific and legal obligations by scientific societies. But we proceeded with the trials as phase I, II, III. But we cannot remain confined in this particular phase forever. Today, after a protracted and ceaseless battle we have reached at a stage of basic understanding by which we can confront cancer in our own way. Day by day the International scenario of advanced medical science is showing some ominous signs as a section of greedy businessmen is investing money in Pharmaceutical industry to get dominance over the technologists and scientists. Mostly they intent to use science for their own monetary benefit rather than the greater benefit of the society. We urge upon the Scientific World to be vigilant about these affairs and look into our research work in right earnest to achieve the desired goal. We pledge that instead of asking for Patent of our “wonder drug” we will keep the transparency of our technology and research works on Cancer for the greater benefit of mankind.

Drug Development Process

Not every compound that is tested in lab is marketed. Before a drug is marketed, it has to undergo several stages of development. A company has to screen through many thousand compounds that show promising result before it could take on the task of development of a promising compound. This eventually increases the cost of development of drug as many compounds that are tested are discarded in the preliminary stages of development. For every 1,000 compounds that are identified by a company, only about 30 show promising results. And for every 30 compounds that show promise, three get past the first round of clinical trials and finally, only one hit the market. Sometimes compounds are to be dropped off during regulatory approval process. Thus, to introduce one new drug, a company needs to start with many thousands of compounds.

It is very known that drug development is very sluggish process which is scrutinized at every stage of development by the USFDA in the US and respective regulatory agencies in various countries. It may take anywhere between 12 to 15 years to develop a new drug according to PHARMA (Pharmaceutical Research and Manufacturers of America - pharma industry trade group of America). This slowness of drug development is attributed to numerous steps a drug has to go through before it is ultimately launched in the market. Drug development includes about six-and-a-half years of discovery, preclinical testing, and toxicity studies; one-and-a-half years in Phase I trials to assess safety in healthy volunteers; then two years in Phase II trials with a few hundred patients to evaluate the drug's effectiveness and side effects. The development process continues with three-and-a-half years in Phase III trials involving thousands of patients and scores of research centers to confirm effectiveness and evaluate long-term effects, then one-and-a-half years of drug authority review, where all the clinical trial data are presented. Even after the drug is approved, it may undergo further Phase IV testing so more safety and efficacy data can be collected.

Drug development in addition to taking longer time for marketing approval (12 to 15 years as discussed above) requires mammoth investment from pharmaceutical companies. It is estimated, according to various sources, that cost of developing a single new drug including commercialization varies from US \$ 800 million to US \$ 1.7 billion [3,4]. Thus, in addition to increasing approval time for a single drug, cost of development is also escalating. This is partly attributed to scrutiny by regulatory agencies and lengthening time for review of applications. It is quite possible that during the stage of development, a drug under review may not make to next stage due to reasons like quality, safety, toxicity or efficacy and thereby increasing the cost of development. The money invested by the company for such unsuccessful molecules is sunk cost and cannot be recovered.

Various Stages of Development of a New Drug:

Preclinical stage:

This stage comprises of study on animals to find out various parameters for a drug under

development. During preclinical drug development, a sponsor evaluates the drug's toxic and pharmacological effects through in vitro and in vivo laboratory animal testing. Genotoxicity screening is performed, as well as investigations on drug absorption and metabolism, the toxicity of the drug's metabolites, and the speed with which the drug and its metabolites are excreted from the body. At the preclinical stage, the FDA will generally ask, at a minimum, that sponsors: (1) develop a pharmacological profile of the drug; (2) determine the acute toxicity of the drug in at least two species of animals, and (3) conduct short-term toxicity studies ranging from 2 weeks to 3 months, depending on the proposed duration of use of the substance in the proposed clinical studies.

Clinical Stages:

Phase I: Phase I studies are carried out in healthy volunteers, which are small in number – usually 20 to 100. The purpose of phase I studies is to identify metabolic and pharmacological effects of drug in humans and to determine the side effects associated with increasing doses, and, if possible, to gain early evidence on effectiveness. During Phase 1, sufficient information about the drug's pharmacokinetics and pharmacological effects is required. The purpose of phase I studies is to mainly determine safety profile.

Phase II: Phase 2 includes the early controlled clinical studies conducted to obtain some preliminary data on the effectiveness of the drug for a particular indication or indications in patients with the disease or condition. This phase of testing also helps to determine the common short-term side effects and risks associated with the drug. Phase 2 studies are typically well-controlled, closely monitored, and conducted in a relatively small number of patients, usually involving several hundred people.

Phase III: Phase 3 studies are expanded controlled and uncontrolled trials. They are performed after preliminary evidence suggesting effectiveness of the drug has been obtained in Phase 2, and are intended to gather the additional information about effectiveness and safety that is needed to evaluate the overall benefit-risk relationship of the drug. Phase 3 studies also provide an adequate basis for extrapolating the results to the general population and transmitting that information in the physician labelling. Phase 3 studies usually include several hundred to several thousand people.

Phase IV: In addition to these three phases, Phase IV, also known as Post Marketing Surveillance is also carried out once the drug is approved and marketed. The aim of Phase IV is to find out safety profile in large patient pool across the world and to establish the safety profile of the drug. It is estimated that success rate of drugs making to market from lab is very less. One drug, from among the thousands tested, makes it to the market.

Cellular Signaling in 3D Tumor Models

Culturing cells in 3D was envisaged decades ago as having potential for use in functional studies of malignant and/or non-malignant tissue. In more recent years, an explosion of new techniques and the extensive characterization of a wide range of cancer cell lines has given

researchers the opportunity to dissect cellular pathways in these more biologically relevant models and, in some instances, make comparative assessments to cells in traditional 2D monolayer culture. If pathways of cells in 2D models are not representative of the *in vivo* microenvironment, then screening for active compounds utilizing these models may not be as predictive. For example, the cellular target of a particular compound may not be expressed in the same quantities *in vivo* or the cellular signaling occurring *in vivo* may not be reflected in cells grown as 2D monolayers and therefore impact on the outcome.

A significant volume of research into molecular alterations of cells has been performed utilizing 3D cancer models, including examination of the genetic variations between different cell culture conditions. In one study, the gene expression of a panel of 24 malignant and non-malignant breast cell lines was compared between 2D monolayer cultures and 3D cell cultures generated on IrECM. Significant modifications in gene expression were detected for genes encoding signal transduction proteins across the panel of cell lines tested, which provides supporting evidence that cellular signaling is altered in 3D cell cultures established on IrECM. In addition, changes in gene expression were also examined in a panel of malignant and non-malignant prostate cell lines. In the 3D cell cultures of these cell lines, the gene expression patterns reflected the decreased cellular propagation upon culturing cells in IrECM in comparison to that of cells in 2D monolayer culture. Furthermore, research into changes in gene expression occurring between 2D and 3D cell cultures has also been completed in melanoma cells. A study demonstrated that 106 genes were up-regulated and 73 genes down-regulated in anchorage-independent 3D cell culture in comparison to the 2D monolayer cell cultures for the same cell line (NA8). The genetic alterations of interest included a number of chemokines (CXCL1, CXCL2 and CXCL3), IL-8 and CCL20 which were significantly upregulated in cells cultured in 3D conditions.

Studies have also investigated specific alterations at the protein level of cells cultured in 3D systems. In a large panel of ovarian cancer cell lines, modifications in cell adhesion marker expression were observed, particularly for vimentin and E-cadherin, in 3D cell cultures when directly compared to 2D monolayer cell cultures. In addition, a proteome analysis between 2D and 3D cell cultures of the colon cancer cell lines COGA-5 and COGA-12 was undertaken. Results demonstrated alternative protein expression of certain proteins, for example, hydroxyprostaglandin dehydrogenase and lamin A/C in COGA-5 cells cultured as spheroids in comparison to the same cells grown in 2D monolayer cultures. A 3D co-culture model for prostate cancer was developed to evaluate interactions between prostate cancer cells and stromal cells derived from the bone. The prostate cancer cell line, PC3, was co-cultured with the bone stromal cell line, HS5, in 3D cell cultures generated on IrECM. In these cocultures, cell-to-cell interactions and cross-talk between the different cell types was demonstrated, with the re-expression of CXCR7 and N-cadherin occurring in HS5 cells. Furthermore, α 6- and/or β 1- integrins were shown to influence the expression of certain cellular components, for example, Ecadherin and vimentin not only in 3D co-culture

conditions, but also in mono-culture of PC3 and HS5 cells. It is evident that culturing cells in 3D alters gene and protein expression, however, it is yet to be determined if any of the changes observed between in tumor cells of 2D and 3D cell cultures will lead to identification of novel drug targets and if the changes in expression alone can influence the sensitivity of anti-cancer drugs. The presence of ECM in the tumor microenvironment has been proven to effect drug activity against a variety of cancer cells. Numerous studies, in a range of different cancer cell lines, have shown that cancer cells cultured in 2D monolayers in the presence of different components of ECM proteins have reduced sensitivity to anti-cancer agents. For instance, in the breast cancer cell line MDA-MB-231, adhesion of $\alpha 5\beta 1$ - and $\alpha 2\beta 1$ -integrin to fibronectin and collagen I, respectively, was protective against paclitaxel cytotoxicity. In lung cancer, a panel of cell lines (H69, H345 and H510) were cultured on collagen IV, laminin or fibronectin and exposed to doxorubicin, cyclophosphamide or etiposide, with cellular attachment to all substrates resulting in increased cellular viability upon application of these apoptotic stimuli. Furthermore, pancreatic cancer cells.

(AsPc-1) cultured on laminin were partially protected from gemcitabine, with the signaling of focal adhesion kinase (FAK) demonstrated to be a contributing factor [53]. Cellular adhesion to ECM has also affected the sensitivity of anti-cancer agents on cells in 2D monolayer culture of prostate cancer and glioblastoma cells. Thus, attachment of tumor cells to specific ECM proteins affects the response of a wide range of cancer cells to therapeutics in 2D monolayer cell culture. The 3D architecture of spheroids, in addition to the presence of ECM proteins in cell culture models, affects cellular responses to chemotherapeutic drugs. A study undertaken by Hakanson et al. demonstrated this concept, revealing that a small 3D structure (less than 6 cells per aggregate) consisting of MCF-7 breast cancer cells was more resistant to paclitaxel in the presence of ECM proteins when compared to the same cells cultured in 2D on a layer of identical ECM proteins. Spheroid models have been utilized to evaluate tumor cell signaling in comparison to 2D monolayer Biology 2014, 3 351 cultures in various cell lines. Notably, signaling downstream from human epidermal growth factor receptor type (Her2) was altered when cancer cells were cultured in anchorage-independent 3D conditions in comparison to a 2D monolayer format. Specifically, a switch from the phosphoinositide 3-kinase (PI3K) pathway to the mitogen activated kinase (MAPK) pathway was demonstrated in breast, lung and ovarian cancer cell lines. The cell culture condition-dependent pathway switch was also observed in a study undertaken by Weigelt et al. using breast cancer cell lines cultured in an IrECM-based model. Studies into the activity of anti-cancer agents against cells cultured as anchorage-independent spheroids have been performed. Mesothelioma cancer cell lines (M28, REN and VAMT) cultured in both 2D culture and a 3D anchorage-independent cell culture format exhibited resistance in the 3D system in response to application of apoptotic stimuli [39]. Certain proteins from the PI3K pathway were identified as having a role in mediating the observed resistance. Furthermore, decreased doxorubicin activity was detected in selected endometrial cancer cell lines cultured as anchorageindependent spheroids in comparison to the same cells cultured in

2D monolayers. The enhanced resistance was potentially associated with cell line-dependent mechanisms including altered signaling through the PI3K pathway and altered antioxidant protein presence. Variations in drug activity against cells cultured as anchorage-independent spheroids compared to the same cells grown in 2D monolayer cultures is not unique to the types of cancer mentioned above. Altered drug sensitivity in cells cultured as anchorage-independent spheroids compared to cells cultured in a monolayer has also been observed in bladder, pancreatic and colon cancer. Altered signaling and sensitivity to anti-cancer agents was also observed in cell lines when cultured as 3D structures using lrECM as a substrate. The susceptibility of breast cancer cell lines over-expressing Her2 (AU565, SKBR3, HCC1569) to therapeutics targeting Her2 signaling (trastuzumab, pertuzumab and lapatinib) was cell line-, cell culture condition (2D vs. 3D)- and drug-dependent. For instance, SKBR3 cells were significantly more resistant, AU565 cells were significantly less resistant and HCC1569 cells displayed an equivalent activity profile to trastuzumab in 3D cell culture in comparison to 2D monolayer cell culture. Furthermore, the results from this study also demonstrated the influence of the surrounding ECM microenvironment on the response of cells in 3D cultures to the Her2-targeted therapies by showing the combination of a β 1-integrin inhibitor with each anti-Her2 agent generally enhanced the anti-tumor activity. 3D modeling employing lrECM in the culture microenvironment has provided a unique tool for use in the elucidation of cellular signaling mediated by integrins. An early study demonstrated that inhibiting the function of β 1-integrin in breast cancer cells (T4-2) cultured in 3D conditions triggered a reversion of these cultures to a non-malignant phenotype. In the same study, α 6- and β 4-integrin function was inhibited in a 3D cultured non-malignant breast cancer cell line (S-1), and following treatment, these cultures exhibited features observed in malignant phenotype. Further research demonstrated that the phenotype of multiple breast cancer cells showed at least a partial morphological reversion to normal tissue architecture when exposed to a number of specific inhibitors applied in combination or as single agents, for example, those targeting MAPK and/or β 1-integrin. Additionally, research was conducted into the influence of integrin binding on the formation of 3D structures. A synthetic hydrogel consisting of RGD binding sites was utilized, with results demonstrating enhanced growth of ovarian cancer cells upon integrin attachment to the substrate.

β 1-integrin was also explored as a potential drug target utilizing 3D cell culture models situated in an lrECM-containing microenvironment. Blocking the function of β 1-integrin in breast cancer cell (T4-2, MDA-MB-231, BT-474, MCF-7 and SKBR3) cultured as pre-formed 3D structures successfully inhibited the growth of these malignant cells and enhanced the anti-cancer effects of breast cancer cells (MCF-7 and T4-2) following exposure to ionizing radiation. The investigation of resistance against anti-Her2 therapeutics in breast cancer cell lines revealed that β 1-integrin downstream signaling has a role in mediating this altered sensitivity. β 1-integrin was also demonstrated to be protective against several anti-cancer agents in hepatoma cells. Furthermore, attachment of cells to lrECM was shown to protect cells from apoptosis in ovarian cancer 3D cultures upon exposure to the PI3K/the mammalian

target of rapamycin (mTOR) inhibitor, BEZ235. The altered drug sensitivity was attributed to the up-regulation of pathways specific to cellular survival. Targeting the pro-survival protein, Bcl-2, insulin growth factor type 1 receptor (IGF1R) or epidermal growth factor receptor (EGFR) in combination with BEZ235 abolished the resistance observed with matrix-attached cells. Therefore, the presence of ECM is an important factor when considering the efficacy of therapeutics in the in vitro 3D tumor microenvironment.

Together, these studies highlight the differing genetic profiles, protein expression and drug sensitivity, which are evident in cells cultured in the more physiologically relevant 3D cell culture models compared to traditional 2D cell culture models. These studies also emphasize the importance of using 3D cell cultures to complete mechanistic studies on current chemotherapeutics and novel drug candidates. An awareness of the differences, sometimes significant, between cells cultured in 2D and 3D is an important factor when considering which model to select for the screening of new molecular entities. The benefits of screening biologically active compounds against cells in 3D culture models is their ability to account for these changes e.g., the switch in PI3K pathway signaling to MAPK pathway signaling observed in 3D cancer cell cultures, but not in 2D monolayer cell cultures as mentioned above. The challenge is to incorporate the essential elements of these models into early stage drug discovery practices.

Utilizing 3D Tumor Models in Drug Discovery: Progress So Far

The development and use of 3D cell cultures in drug discovery is becoming more prevalent. At the present time, a collaboration of academic laboratories and pharmaceutical/biotechnology companies in Europe has been established to develop new, more relevant, in vitro models for drug discovery practices. Numerous methodologies have been established for novel compound screening practices utilizing 3D cell culture systems in cancer, particularly within the last few years. These procedures have included both non-adherent 3D cell cultures (anchorage-independent) and 3D structures which adhere to a substrate (anchorage-dependent).

Numerous anchorage-dependent models for drug discovery have been developed. Recently, developed miniaturized 3D cell culture assays utilizing small panels of both breast (MCF-7, MDAMB- 231 and BT-474) and pancreatic (Panc-1, AsPc-1 and BxPc-3) cancer cell lines, suitable for use in drug discovery programmes. The assays established were based on 3D cellular structures situated on IrECM in a 384-well microtitre plate format, a configuration compatible with a range of liquid handling, imaging and multilabel plate reading equipment.

Research Uses for Mice:

The most common application of mice in cancer research is to test in an in vivo setting the hypotheses about cancer biology and physiology that investigators generate in cell culture studies or from clinical observations. More recently, cancer researchers employ mouse models and inbred strains to explore their applicability to projects that identify novel

potential targets for therapy or prevention, to test new clinical agents, and to understand the genes and environmental factors that contribute to cancer susceptibility.

Aspects of basic biology and physiology of both normal functions and malfunctions in mice yield important clues that translate to human biology. Examples of many types of mouse models that deliver data on genes and pathways to provide insights and research directions are available. Testing experimental therapeutics in pre-clinical or co-clinical settings is crucial for many reasons. Efficacy of potential treatments can be explored quickly in mouse screens. Safety or toxicity can be examined. Better mouse models can be developed as we learn about which features of a treatment are most parallel to situations observed in human treatments. Prevention research that could offer guidance on ways to postpone or eliminate cancer would have tremendous value for public health. Insights into the genetics of susceptibility and risk factors, paired with prevention strategies, will benefit us on many fronts.

Research Uses for Rats

Rats have been used for decades to learn more about the causes, the biology, and the treatment of cancer using a variety of methods. One of the more common applications of rats in cancer research is to screen compounds for toxic effects and resulting cancers in large-scale studies. A number of these studies are underway, and have been providing insights for years on the affects of chemicals and environmental assaults on the animals. Aspects of basic biology and physiology of both normal functions and malfunctions in rats yield important clues that translate to human biology. One of the earliest realizations of the genetic nature of cancer was provided by the Eker rat model, and subsequent work with that model has continued to provide knowledge about the mechanisms of cancer.

Experimental therapeutics is frequently tested in rats to evaluate efficacy, safety or toxicity. Because of their larger size, rats are also the rodent of choice for interventions and studies that involve surgical treatments. Improved drug delivery strategies, and advances in imaging strategies that will help clinicians to better understand the images obtained from patients' tumor assessments, are also demonstrating the utility of rats as a model to benefit human health.

Prevention research that could offer guidance on ways to postpone or eliminate cancer would have tremendous value for public health. Research into the genetics of susceptibility and risk factors, paired with prevention strategies, can be performed in rat model systems and yields new knowledge every day.

Other lab animal models

For various reasons, some researchers pursue knowledge on the biology and physiology of cells and tumors in models that have features that are different from those of rats and mice. For some models there are benefits of size, historical framework, and tractable features that support the routes of inquiry more readily. These include rabbits, hamsters, and zebrafish that are regularly used in laboratory investigations of cancer.

SEARCH FOR AN EASY WAY TO TREAT CANCER (EPISODE 1)

The year 2020 will face cancer as an epidemic – as informed by world Cancer Society. Yet we do not possess any medicine to cure it. A population of about one and a half crores may be attacked by this disease by the next decade. Population of countries like India, Bangladesh, and Pakistan may increasingly be affected. We get all these information from International Medical Journals.

Dr. Chatterjee had spent three long decades to research on treating and curing all types of cancer and still continues with the process. He is the one to inform us about the expensive treatment of cancer. 85% of India's population fails to afford it. People with the income of 40-50 thousand per month can only undergo the conventional process of treatment, i.e. only 5% of the population can afford the treatment.

Besides, Dr. Chatterjee also makes us aware of the fact that India most often fails to detect cancer in time. Some arrangements have been made up only in urban areas. In that case, in India, mostly the disease is detected at a much later stage and as such doctors are also helpless. The weak public health system is responsible for this inadequate, yet expensive arrangement to treat cancer. But he informs that oral cancer is mostly prevalent in India. So we should always avoid tobacco products. Dr. Asim Chatterjee further informs that he keeps on trying Psorinum therapy on cancer patients since long, though it's still limited within several experiments. This therapy, along with the necessary palliative treatment has produced some positive results. According to him, India may play a dominant role in future in treatment of cancer with the help of this therapy. Families with lesser income should be able to undergo treatment of cancer provided we work in this direction. To achieve this goal Dr. Chattejee has reoriented his own residence and has set up a model Cancer research and treatment centre.

There are different side effects of cancer. As such the human body loses resistance power, infection spreads, excretion is disturbed, indigestion increases, small particles of blood decreases, respiratory problems prevail. In that case, saline, antibiotics, rice-tubes, surgery are common as palliative management in order to remove the obstructions of the patient body Psorinum therapy is one type of medicine which is consumed orally. It does not have any side-effects.

SEARCH FOR AN EASY WAY TO TREAT CANCER (EPISODE 2)

Critical Cancer Management Research Centre and Clinic, in Lake Town, Kolkata, has experienced tremendous success in both palliative treatment and Psorinum therapy for cancer patients since last three years – this information percolates through Dr. Asim Chatterjee. But he also admits that India is much behind the western countries in treating the disease because this is detected at a much later stage in this country. If detected at an early stage India is also able to treat cancer to a greater extent.

Dr. Chatterjee was asked about the way in which this ‘Critical Cancer Management Research Centre and Clinic’ could provide for easily accessible way of treating cancer. He told us cancer is around 120 types. Each type produces different reaction on human bodies. Moreover, cancer yet does not have any medicine. We do not have any other ways except Chemotherapy, Radiation and Surgery. These are indeed expensive. 85% of Indian population fails to afford these. Patients and their families become helpless at different situations. Dr. Chatterjee further says that he continues with his research on medicines since last thirty years. Moreover, there is always an attempt to stand besides the helpless patient’s families. Because he feels this is a problem of the entire society. We should all come forward together to fight cancer.

How did you set up a terminal centre for Cancer? ‘Actually, my friends, Sri Tapan Sen and Sri Shankar Chakraborty have encouraged and cooperated in making this institution. My first patient in this institution was Shankar da’s elder brother Sri Kanai Chakraborty. Shankar Chakraborty’s contribution in building up the infrastructure for today’s system of Cancer palliative treatment is praiseworthy and commendable. At present, my son, Sri Aradeep Chatterjee is working in joint collaboration with some institutions abroad on the effectiveness of this drug of Psorinum and to find out its molecule.’

Dr. Chatterjee, himself intended to go to U.S.A for his work. “Actually one of those who taught me the details of cancer has lucidly explained me the fact that a country’s problem should always be dealt within the soil, infrastructure and apparatus of that country only, in order to make the work valuable to the general mass of people. Today, our centre has been recognized as one of research and treatment of cancer by American Society of Clinical Oncology.”

Regular counselling is provided to the concerned patient and his/her family. Even from 2007, there has been an attempt to provide the patient’s families coming from outside Kolkata with cheaper arrangements to stay in Lake Town with the help of Gandhi Seva Sangha. Dr. Hiranmay Saha, Manju Mukherjee, Goutam Saha, Jaba Guhathakurata, Raghunath Kundu and Mani Chakraborty have also contributed a lot.

SEARCH FOR AN EASY WAY TO TREAT CANCER (EPISODE 3)

“We need to understand the treatment of cancer, the concerned patient and the problems of patient family through a panoramic view. It should be clear to us that this problem cannot be encountered only by state initiative, doctors, nurses, medical scientists, pathologists, or one or more than one social organizations. Instead, 120 crores of people comprising whole of Indian population should come forward to realize the depth of the problem and this problem should be treated as that of all” – said Dr. Chatterjee.

Even if detected at an early stage, cancer treatment is centered on some technology based processes like Surgery, Radiotherapy, Chemotherapy, Immunotherapy etc. These are indeed expensive. Many families in India try hard to save their patients at the cost of all their belongings, but if the disease is not detected at an early stage. All efforts will be futile. The family will be totally ruined. Instead of surgery, sometimes Chemotherapy is applied to the cancer patients on the basis of biopsy report which denotes the Karnofsky scale of the health condition or medical status of the patient. If the medical status is below 50 of the Karnofsky scale then CT is not applicable, as it may show some side effects in the patient's body. It kills both the ordinary as well as the cancer cells of the body. This may cause hair fall and loss of immunity and other health problems together with hair fall. In these days, in case of bone cancer, an injection viz zoledronic acid is needed to keep the bones strong. Bone scan and blood tests are urgent before this injection is applied. Moreover this injection cannot be given without the help of cancer specialists and oncologists. It has a dose for 6 months, once in a month. It costs above Rs. 6000. A patient has to sit 6 hrs after the acid is injected. It is important to note that scanning of the bones of the entire body is not possible everywhere. Generally this kind of a treatment is beyond the reach of economically backward section of the population in both rural and urban areas. The injection has of course given some positive result but if cancer spreads in the entire body then things become difficult.

Dr. Chatterjee has informed that since few years, they have been applying a special Psorinum Therapy instead of Chemotherapy, where affected portions are lungs, liver, stomach, gall-bladder, pancreas, internal parts of mouth etc. This therapy is of course applied on the basis of biopsy report and simultaneously with the conventional treatment. It produces positive results in many cases, by expanding the life-span of a patient. The reason behind the recognition of Dr. Chatterjee's institution 'Critical Cancer Management Research and Clinic' as an unit of both research and treatment by American Society for Clinical Oncology, is due to the experimental success of Psorinum therapy. Besides, some scientific papers have been published on Psorinum therapy. Today, the scientific world has accepted the Psorinum therapy as a treatment of cancer. The experiments are still on. Our aim is to build up the institution without government grant. It is gratifying to note that this kind of initiative only

for cancer patients is very rare in India.

Different social and financial limitations are closely associated with cancer treatment. People from distances come to Kolkata very often to treat cancer. Dr. Chatterjee says that he is aware of the difficulties of the people coming from outside to stay in Kolkata for treatment. So he is trying to make arrangements for their stay at Kolkata for a temporary period. Initially, there were some difficulties. But with the cooperation of local inhabitants of Lake Town some arrangements could be made through the Gandhi Seva Sangha from 2007.

Dr. Chatterjee reiterates, I do feel the need to develop system to treat cancer at an early stage at the regional level. We should be aware of the increasing number of cancer patients. So we should consciously come forward to encounter this problem and treat the disease within their affordability with the guidance of the specialists. Only government initiative would not be enough to fight this situation.

(Source: Interview earlier published in Bengali for the monthly newspaper Sangbadmanthan, dated, 16th May, 2011, Dr. Asim Chatterjee's interview by Sriman Chakraborty, Kolkata)

A TODDLER'S HOP FOR RETRIEVAL OF LIFE FROM CANCER

The Crawl To Reach Out

I have been actively working on Cancer for long 35 years or even more. For any person, when he gets engaged into any research, he has to step-by-step acquire complete knowledge regarding the matter under study. This happens in most of the cases. And I am no exception to it. Our country, India, is lagging behind in most of the socio-welfare research or social-wellbeing-related activities, the major reason behind this being lack of opportunity to the most of the deserving people. But in developed countries, a child gets ample support with timely guidance, and his interests are properly looked after. As a result, they love what they do and ultimately they emerge as eminent and passionate personalities. Here, I would like to talk about a Pakistani singer, Noorjahan, who, when asked by a journalist on how to become a famous singer, replied, "It is a blessing. The talent needs to be nourished and the skill needs to be developed to become recognized."

My journey to combat Cancer has been going on since 1974. My elder sister's demise because of cancer made me resolve that I must have to cross a long dark road in order to throw new lights of knowledge on that deadly disease. I began to find out an alternative way to the prevailing complicated method of treatment of Cancer, but being a student of science I did not know much about Sanskrit, the language, which deals with Ayurveda in details. In that case, homoeopathy came as the only choice. Having been well-acquainted with homoeopathic treatment, I knew that it had serious limitations. It is neither wholly symptomatic nor also based on any pathology. Even modern school of thoughts in this field believe that no scientific work is possible without any new molecule in the substance referred to as a medicine. When I started my work on Cancer, I had a very vague knowledge about the subject. In the previous volume, "A Total Strategy Against Cancer", I have shown how I had observed the cases, gained knowledge, and step by step became able to reach the nucleus of the subject.

It is my misfortune that during the early days of my life, as I inherited a lot of family wealth, I wasted my most valuable time. But it is also true that the coming days will certainly prove whether those times were really wasted or my destiny had something else in store for me. When I started my research, at that time along with various other woes in human life, pain of Cancer-patients and their tragic fate grabbed my attention the most. I did not want to limit myself only to medicine, hospital, government recognition or any awards. Rather I tried all the time to bring the entire problem to one single point of accessible and sustainable solution.

My Preparations For The Venture

DECISION PERIOD, STUDY PERIOD, WORK PERIOD AND WAR PERIOD

DECISION PERIOD :

For almost five years i.e. from 1980 – 1985, I got myself fully engaged in the primary study of basic allopathic treatment of Cancer under the guidance and supervision of Prof. R. S. Bhakta and Dr. S. R. Sikdar. In those five years I gathered a basic idea about the disease, its treatment, allopathic medicine, its system and also effectiveness. Prof. R.S. Bhakta and Dr. Sikdar suggested that I need to have an institutional study on this subject. By this time I could also convince Prof. R. N. Brahmachari to allow me work with him. This time can be termed as the ‘**Decision Making Period**’ or ‘**Incubation Period**’.

STUDY PERIOD:

The ‘**Decision Making Period**’ was followed by the ‘**Study Period**’ which was from 1985 – 90 when I was working with Prof. R. N. Brahmachari and Prof. Anup Majumdar at Cure Point Nursing Home. Here I got to know more about Cancer, the treatment methods like Chemotherapy, Radiotherapy and surgery. I had to put in a lot of efforts personally towards this. Sometimes I hardly found time for full sleep, timely meals or rest. From Prof. R. N. Brahmachari, Prof. R. S. Bhakta and Prof. A. Majumdar (surgeon), the then Director of SSKM Hospital, I acquired the basic knowledge on surgical treatment of the disease and by 1990 I had an overall idea on the totality of the subject. Prof. R. N. Brahmachari in various ways used to fill in the gaps that existed in my perception.



At Cure Point Nursing Home

This was the time Prof. R. N. Brahmachari, Prof. R. S. Bhakta, Prof. Majumdar (surgeon, & former Director of SSKM Hospital) and Prof. Anup Majumdar (former Professor of Radiotherapy, Radiotherapy Dept. B. S. Medical College, Bankura) felt the need to involve the teaching institutes in my study. I required to get acquainted with the hospital infrastructure, and had also the need to see more number of Cancer patients which was not possible with the limited resources of the nursing home. The challenges we were facing while working in the nursing home were the constraints of basic knowledge while the infrastructure was also not sufficient to support such a lofty vision. The requirement of a medical training

institute arose in order to have access to many patients under one roof, to gather knowledge on the core subjects of medical science and also to cater to the need of the combination of treatment, education and research, which was possible to obtain only in a medical college-hospital. However, Prof. R. N. Brahmachari once said: *In reality it is going to be very difficult as you lack the eligibility or qualification that is required to perform this kind of studies.* But he further added: *You have a very curious mind. You want to know everything but unfortunately, you do not come from a medical background. You are not an MBBS student and that acts as a constraint. Now it is not possible for you to get the MBBS-degree but you have to complete the course of both MBBS as well as MD.* I acknowledged this unkind truth. To this Prof. R. N. Brahmachari explained : *The field of education is not a battle-field. No war exists in the sphere of knowledge. We are here preparing ourselves to declare a war against Cancer, and as we all know that there is no border or rule followed in a war. There you either win or die. In our case win is not certain, but definitely death would be a lot easier. Again I repeat that there is no place for war in a students' life. There have been instances in the field of medical science all over the world where professionals have overcome several barriers and emerged victorious.* He then gave me a book to read, authored by Dr. Manish Pradhan and titled “Behind the Discovery” to understand things a lot better. The words uttered by Dr. Brahmachari left a lasting impact in my mind. I read the book again and again.



Cure Point Nursing Home



Prof. R. S. Bhakta with Dr. Asim Chatterjee



Prof. R. N. Brahmachary with Dr. Asim Chatterjee

The people I started up my work with as a beginner were all very prominent personalities in the medical community. They used to restrain me on many occasions. There were a few works that I had to do, which were beyond the limits of medical laws and which they declined to approve. Prof. Bhakta had also once objected to my activities. For almost one year we had no communication with each other. He often used to ask as to why I was always indulged in these kinds of work. To this I clearly stated that I was neither a physician nor a scientist. My work is to create a new nucleus which would be beneficial for the entire humanity.

WORK PERIOD :

The '**Work Period**' from 1990 onwards kept me mostly engaged:

1. To gain access to the platforms of scientific research and development like SSKM Hospital, School of Tropical Medicine, Chittaranjan National Cancer Institute (CNCI), Radiotherapy department of Medical College, the Indian Association for the Cultivation of Science and the Tata Memorial Hospital.
2. To convince a few stalwarts like Prof. Subir Dutta, Dr. Saroj Gupta, Dr. Gauripada Dutta, Prof. Amar Bhaduri, Dr. Amar Bhaduri, Dr. Dipankar Dasgupta, Prof. Sisir Kumar Dutta, Prof. A. K. Hati and a few more.
3. To overcome several critical situations at certain points of time through several processes including the creation of a few NGOs.
4. To publish research papers with my fellow colleagues and to overcome many financial and legal hurdles at various points of time.
5. To join Subodh Mitra Cancer Hospital with a view to experience how to administer a hospital efficiently and effectively.
6. To fulfil my social commitment, taking of steps for the rehabilitation programs for the patients and a shelter for their family members who used to come from distant places

during my tenure at Gandhi Seva Sangha.

7. To enter international platforms like American Society for Clinical Oncology, NCI (National Cancer Institute, USA) for publishing papers and articles in many international journals etc., used to be completely taken care of by my son, Dr. Aradeep Chatterjee.
8. To perform the drug-trial on a large number of patients in order to move ahead with my scientific research and towards successful development of the drug.

In this perspective, I made the dive to a sky not explored much.

WAR-PERIOD:

In the initial days I faced several problems as I did not have any degree in Radiotherapy, Physiology or Biochemistry (neither MD nor M. Sc.). During my tenure in the Indian Association for the Cultivation of Science, Cure Point Nursing Home and my association with some renowned personalities, I had learnt how to go about my work and also gained the experience of working with eminent scientists of those times. I also learnt the importance of the documentation and publication of research works. But soon I realised that I am meant to do something that would be beyond anyone's imagination. After coming out from the Indian Association for the Cultivation of Science I chose to work on cancer of lung, liver, gall bladder, stomach and pancrea. Simultaneously I was also working on blood cancer (AML) and brain tumour (GBM), renal-cell carcinoma and sarcoma. The patients that I chose were lying below 50 in the Karnofsky scale. Modern research has not yet been successful to treat these cases. So I strategically took only those patients who were proper institutional cases. The institutions that I targeted then were Chittaranjan National Cancer Institute, SSKM Hospital, Thakurpukur Cancer Hospital, Medical College Hospital, Dr. R. Ahmed Dental College and the School of Tropical Medicine. I thought if I could treat these institutional cases and help in the regression of the disease, by extending their life for a few more years, I would secure a position for myself and I would not have to face any legal consequences, which was quite often then. In that situation the scientific world might also show some acceptance towards me.

My strategy was to convince and treat those patients who were initially rejected by the hospitals on the ground that their cancer could not be treated anymore and the patients had only a few days or weeks left to survive. I treated mostly these patients and after a certain interval of time took them to those respective hospitals for their check up and then to document their current medical condition. This was a very difficult task and while walking on this path I had to make friends with all sorts of people and often I had to gratify them also in their desired manner. One cannot aspire to achieve the goal with the right intention without taking certain harsh and morally challenging decisions. However, I was able to document many of such cases in this way.

Gradually I also convinced and got access to the Pathology Forum, Radiology Forum and the Oncology Forum. This period went on till the end of 1990s. Within that time my work received all the deserved recognition on both national and international platforms. I was happy when the scientific forum expressed their acceptance towards me.

I never wanted to become a scientist, though I had the option to become such, working

in the Indian Association for the Cultivation of Science and later in the School of Tropical Medicine, but I wanted to serve the humanity staying amidst the needy than within the four walls of the laboratory. But now after so many years I feel that it was equally important to work on the drug's pharmaco-kinetics and pharmaco-dynamics. Then only it might have opened new avenues for the treatment of the disease.

While going through this the reader needs to keep in mind that I was neither an Oncologist nor an Onco-scientist. But from the very beginning I knew that I would have to become one of them. I felt the need to get enrolled in some teaching institution and continue with my studies and undergo some kind of institutional evaluation at the same time. But this was not possible as I did not come from a sound medical background and I did not possess the required qualification. I wanted to see more and more patients and get acquainted with the situation and also with the ongoing treatment methodology, and that would have been possible only in renowned hospitals. I got the first taste of this in Cure Point Nursing Home, and after that my hunger rather increased. I felt just like a tiger that has tasted blood and is looking for more.

I strategically came in direct confrontation with some renowned hospitals and eminent personalities. I received some psychological satisfaction during my tenure at the School of Tropical Medicine and then at the Medical College Hospital, Kolkata. Very soon I became familiar to the entire treatment, management as well as administrative part of the institutions concerned. I read and at the same time observed that there are certain types of cancer which are resistant to Chemotherapy and Radiotherapy. Again if the case falls below 50 in the Karnofsky scale, Chemotherapy and Radiotherapy cannot be administered. Surgery is also not an appropriate option in certain cases. I decided to work with these kinds of end-stage cancer patients and by God's grace, condition of a few of them improved, which was nothing less than a miracle. Irrespective of all this I had to face many physically challenging situations and indulge in many table-fights to win the confidence of my contemporaries and at the same time the patients who required my immediate help.

Initially I had to face severe resistance from the IMA, Pathology Forum, Radiology Forum and specially the Oncology Forum. Eventually there was truce between us. We realised that we will have to forget our enmity for the greater good and need to move forward together, holding each other's hands. I have presented a few of my research papers here which I have written in association with some renowned figures from different spheres of the medical field. I have also mentioned how the NGOs helped me overcome certain critical situations. Since it is not possible to furnish here all the details of my work and journey so far, I have kept aside some material which I desire to bring out in the next book.

The Cancer-Treatment Scenario In 1980S And My Journey

As I have already mentioned, I started my core research on Cancer from 1980. That was the time when the western countries just introduced some technological methods to the world of medical science in order to detect Cancer i.e. through pathology. These included USG and C.T. Scan. However, only a few government hospitals and private nursing homes in India

could manage an access to these technologies much later. Thus, technology-based detection of Cancer started in metropolitan cities of India only from 1990s. These were accompanied with the costly methods like radiation and chemotherapy. But due to absence of any effective medicine these advanced techniques did not produce much fruitful results. This shows that the centuries-old highly technical infrastructure of medical science is still inadequate and incomplete, so far as a wholesome approach to cancer-treatment is concerned.

The initial days of medical education and practise did not have any Medical Council, Scientific Academy or University to monitor the research works. From 16th century onwards, we have the British Royal Society, French Royal Society etc. In 20th Century striking improvement was noticed in the medical technology. Medical Council, Scientific Forum, Medical College and Universities were built up as much-needed infrastructure for the modern world of medical science. Their contribution cannot be denied. Still then, there lies a big gap in the process of reaching the desired goal from a practical aspect. In Third World countries or in countries under colonial domination people are not often treated fairly. Their research work, if any, did hardly get any legitimate recognition and respect, and used to be often looked down upon by the advanced countries. Even the literary creations and genuine research works of our country are not duly appreciated unless and until they are recognized by the so-called developed countries. At the same time the developed countries by and large have in possession the required infrastructure to carry forward any pioneering project of research. Besides, they are also provided by the concerned authorities with both cooperation and encouragement required to promote new and pioneering research projects.

It is strongly felt that a person needs to be an inhabitant of his own country in order to understand the specific problems of that country. He must have an understanding of sufferings of his own people. Sometimes national or international organizations come forward to deal with some issues. But it's not always possible for them to realize all the internal problems of an alien country or society. Moreover the local people often hesitate to interact with them. Over and above, a person has to work within the limitations of the country's judicial system and the social fabric of such country. Initially no original works blossom within any prescribed or pre-specified frame, and can be butted or bounded by any set of too rigid rules and regulations. But later, things need to be set in a proper framework in a scientific and logical order. The irony of fate is that even after fulfilling all such paraphernalia, such research works hardly stand the acid test of authoritative recognition until and unless duly acknowledged by the European and the American countries or by any international journal.

In 1980 what was the status of Cancer treatment in Calcutta? With a practical reference to that, how did someone who is not a scientist could dare to begin his research work! Though "New Resource", an NGO newly formed, provided me with some non-technical facilities, still what about the technical ones? There were so many hurdles which I had to cross over. I came to my present residence at 381, S.K. Dev Road, Kolkata — 48, from Basirhat in the suburb of Kolkata not long before. So primarily I needed a social recognition as an inhabitant in the

locality. They did not even know me properly. What I needed initially was to reach out a few Cancer patients, and to make arrangement for their accommodation and for their curative treatment. But it was hard for me to get any access to tissue-proven or biopsy-proven Cancer patients for such study or treatment. There was almost no MRI or CT scan nearby in 1987. At present, however, the scientific world identifies Cancer patients even without tissue proof, just on the basis of MRI or CT scan reports. But this was not possible at that time. Tissue-proof was the only way to detect Cancer for medico-scientific purposes. What legal right did I have to carry on my indigenous treatment procedure? After long and extensive deliberations in the New Resource as well as with many others, we shaped our strategy to treat the destitute and terminal-stage Cancer patients only. They were the patients usually with fragile health and unfit for conventional therapy. I used to observe the treatments of Cancer patients in different private and government hospitals as an outsider. I did not have close association with any doctor; still I used to search for Cancer patients mainly from government hospitals. I built up contacts even with agents staying near such hospitals and also grew quite friendly terms with lower staff of the hospitals. In this way I used to collect information about Cancer patients, and shortlist those for my research work.

This was the time when I was also to some extent engaged in politics, which helped me a lot. 1988–1994 was the time when many hospitals used to be surrounded by various types of agents and middlemen. These people used to dominate most of the sensitive and important areas of medical treatment in Kolkata, like admission of patients, arrangements for stay of patient-families as well as for provision of food and medicines for patients. They used to be found often engaged in alcohol consumption around the hospital areas. In such circumstances it was really difficult sometimes for the authorities to effectively look after the management of hospitals and the process of addressing all minute problems of the patients and redressal thereof. We tried to know about Cancer patients mostly of government hospitals for obvious reasons, as the administration in such hospitals used to be little liberal. We tried to get in touch of suspected cancer-patients through such agents, though there was no financial interest from either side. The agents and we could sometimes meet with each other at tea shops only. Our objectives were very specific i.e. to render effective help to Cancer patients.

What a pitiable kind of infrastructure was usually provided to Cancer patients at that time! When the patients were detected with the attack of Cancer and the location of the disease in the patient's body, it was my primary duty to know how such patients were being usually treated at the hospitals and at what stage of the disease. If I could know their conditions, then only I would go to talk to the patients' families, although I did not have any legal right or obligation to provide any medicine over and above the hospital prescription.

1987–88 was the time when I became more determined to work on Cancer and as mentioned earlier, my working experiences with my ultra-political friends of 1970s taught me to utilize the people of grass-root level (whom we bluntly called anti-socials) in my own work. Thus I renewed my connections with them, with the agents and the menial staff of

hospitals like SSKM, Chittaranjan Cancer Hospital, and Calcutta Medical College. I, with great difficulty, managed to convince many patients to use my medicines but due to lack of experiences I often failed to manage the entire show.

1988 witnessed quite poor infrastructure of hospitals in Kolkata. A number of patients used to get admissions in hospitals in exchange of money; food for patients was often stolen; business of alcoholic products around hospital areas was rampant, and political opposition groups were hyperactive. These issues were of unimaginable gravity. Cancer institutions as usual were guided by the Western protocol. There was no tissue-proven Cancer patient then. We only dealt with patients affected with lung, liver, gall bladder, stomach and pancreas. Then biopsy of lung Cancer meant only taking out the tumour by a hard tube. But this method was very difficult and strenuous. Again there used to be huge confusion between tuberculosis and lung Cancer. There was no CT scan as such; x-ray was not enough to detect Cancer. Besides, when at times government hospitals could detect Cancer, there used to be much hurry on their part or on the part of the patient's family to treat the patient and administer all strong medicines on them. This is not what I was looking out for.

As a result I had to face both physical and mental harassments. But situation got changed from 1990s. Hiding my identity I continued to visit different wards and meet different doctors. Initially doctors were not so responsive and confident about me but gradually their ideas changed. I was provided with case-histories of some registered patients of those hospitals to try my Psorinum therapy on them, and in some cases I indeed achieved miraculous result. These cases involved patients like Smt. Parul Bala Dey (female; lung Cancer case; Chittaranjan National Cancer Institute), Bijoy Kumar Hui (stomach Cancer, Chittaranjan National Cancer Institute), Binapani Sarkar and Sujit Kr. Paul (Thakurpukur Cancer Hospital), Nanda Rani Banerjee and Gobindo Das Ghosh (Calcutta Medical College Hospital), S. P. Deb Roy (SSKM Hospital, Kolkata, colon, liver Cancer, metastasis of lung), and Gautam Paul (Tata Memorial Hospital, Bombay). Their conditions remarkably improved through my Psorinum therapy. These patients were even taken back to the hospitals to register their improvement, inviting thereby government recognition. Huge amount of money used to be spent to treat these patients by their families, except for the Psorinum Therapy.



Chittaranjan National Cancer Institute, Kolkata



Calcutta Medical College

In such a situation, a lot of mental strength was felt necessary to undertake such an against the-flow sailing and to carry it forward. The difficulty was very much there due to absence of proper infrastructure. Thus those who were working for my new research project needed to have firm determination and social responsibility. Several ways had to be negotiated to utilize the scattered materials available here and there in order to reach the ultimate destination. Many of us may not have the mental, physical or organizational strength required to cross such hurdles. Perhaps that's why personalities like Dr. Subhash Mukhopadhyay (inventor of test tube baby) had to commit suicide. To sum up, I was trying to innovate a new therapy, namely, Psorinum Therapy and was deadly out to experiment its application mainly on terminal type cancer patients at a nominal cost.

It is very important to note how at the beginning of my journey I came across various people who had already left an impression in their respective fields. I had to convince them in different ways and I always tried to bring them on the same board since I always had the perception that this work could not be done alone. I needed to have people accompanying me, standing by my side and supporting me. I have always asked for their consent in the decisions that I took and I also did consider and appreciate the suggestions that eventually came up. Here I would like to give a brief note on how I convinced them and the subsequent chapters would describe the circumstances I had to sail through.

Again previously nobody ever thought of working with Psorinum Therapy as far as my knowledge is concerned. This gave me the opportunity to research more on this subject without anyone pointing a finger at me. During 1994 – 97 my papers were presented in many seminars and also in the abstract of National and State Conferences, which were later fully published in the School of Tropical Medicine's Medical Bulletin as well as in a Bengali paper published by the Directorate of Science and Technology.

During my tenure in the Indian Association for the Cultivation of Science, also at Medical College, SSKM Hospital (PG), School of Tropical Medicine, Tata Memorial Centre for Cancer Research, Bombay (now Mumbai) as well as in some other institutions I had observed a significant difference between Cancer and any other disease. Cancer is not a bacteria or

virus. It is an abnormal growth of our body cells, and this multiplication of cells can occur in any part of the body and can affect any vital organ. Different organs of the body have different mechanism, and if infected with Cancer they exhibit different problems associated with that.

For more than 200 years professionals have been working on Cancer, its research and treatment. Significant improvement has been observed after the Second World War. With the advancement of science and technology the treatment methods are also developing, but considering everything together, the process is felt losing simplicity and actually getting more and more complicated. By modern treatment we primarily focus on Chemotherapy, Radiotherapy and surgery. While working on cancer, I felt that a uniform opinion would be required on these subjects for future guidance. There is ample scope for new research because it is our primary objective. We have to have a clear concept of a net result to be obtained from the research activity. Our challenge is to bring our hypotheses to reality. It is very important to match our research result with the conventional treatment mechanism, though it is very tough work. At times it becomes very difficult to perform clinical trials on humans. There are many legal issues involved as well as social obstructions. Again it is very important to keep a track of the ongoing developments and also its documentation is a very tedious process. So there was a need to create a nucleus of this totality.

What I had in my mind in 1980s have been carried down so far with me in bits and pieces and I have tried to give a brief outline of all those in my paper viz. “The Management of Cancer in Totality – India can take a lead.” When it was published, except for a few people, hardly anybody understood my views. The people very near to me advised me to work more on the drug and not to waste time otherwise. They said, I need to work more on scabies and its molecule, and what effect it has on human body. But now, after I have reached this platform with so much difficulty it makes me think that discovery of a proper or single wholesome drug exclusively for Cancer is next to impossible. The reason, inter alia, is that TB, typhoid and other viruses are of one type, while Cancer is terribly different and has multiple facets. Cancer stays and grows as a part of our body and hence it also has some right over our body. Our body also does not deny its rights and gives it opportunity to grow and spread just like a black sheep in the family, who cannot be thrown out but needs to be minded or managed.

However, the medicine, Psorinum, is primarily used to treat certain skin diseases. But I have succeeded in utilizing it to treat cancer, which is proven by pathology. Many people have asked me time and again whether I had to work a lot on this drug but I reply by saying that I had to put in more effort rather in understanding the basic allopathic treatment and to dovetail the effectiveness of Chemotherapy, Radiotherapy and surgery with my treatment methodology. I also had to understand the subjects like Pathology and Radiology and how this helps in detecting the general condition of the patient and in deciding upon the treatment method to be followed in due course.

My Days With The Indian Association For Cultivation Of Science, Kolkata

When I started working on Cancer treatment I had neither any knowledge regarding the

subject, nor I was a doctor. I was told that only an MD in Radiotherapy can clinically detect and treat Cancer patients. It became clear to me that I would have to struggle a lot and also to study Cancer in a way that was well beyond the stereotype. Within myself I took a firm resolve to go ahead. I enrolled myself as a student in the Indian Association for the Cultivation of Science at Kolkata. In my long journey without the conventional knowledge of the subject and without any proper guidance, I had to overcome many barriers and unlock many doors all of which together could pave the desired way to my research and all such challenges encouraged me from time to time to work even more hard and with more determination on this front.

When I was working at Cure Point Nursing Home I got myself introduced to the famous scientist, Prof. I. B. Chatterjee (Prof. Indubhusan Chatterjee). I kept regular contact with him. He had once told me: *There exist many loopholes in your knowledge and I am surprised to know that you are eyeing something which is advanced science. Although I am not a clinician still I can see how well you have delivered the clinical trials of the drug and how documented your clinical data are. I also do trust your genuine works because you are the first person who has achieved to totally regress cancer. But now you have a long way to go. Your subconscious scientific mind wants to do so many things at a time but you are not finding the correct way. I want you to often come to me and understand a few basic things.* Therefore we used to discuss on various things like: What the science is? What is the drug's Pharmaco-kinetics and Pharmaco-dynamics? How to perform animal trials? How to write a scientific paper? What is an index journal? What is cell-line? And no end to it.



Dr. Asim Chatterjee and Prof. I. B. Chatterjee



The house of science and knowledge (Prof. I.B. Chatterjee's residence)



Prof. I.B. Chatterjee



From right to left Prof. I. B. Chatterjee, Dr. Asim Chatterjee and sons of Prof. I.B. Chatterjee

One day Prof. Chatterjee invited me to his laboratory at Ballygunge Science College. There he had also invited many famous scientific personalities of that time. He also picked me up from my residence that day, and in that event introduced me to many renowned people. He then asked me to express my thoughts in front of them. At that point of time I thought that my presence might be inappropriate there. I started off by saying: ***I don't know what to say right now. I have done little bit of work and I would really appreciate if you kindly look into my work and study the details. I have treated three clinical trial cases; one is a lung cancer patient, another liver cancer patient and then an oral cancer patient, and thankfully***

to the Almighty, their cancer has regressed. Apart from this I have not done much work on the subject and I also do not know what I should do next. The people there started smiling and clapping and invited me to join them for a cup of tea. They told me that I had done something that many scientists wanted to do for years together. Again they said: *You have already proceeded with your work and gradually you would learn science as well.* Prof. Chatterjee invited me to visit his laboratory and office frequently and study the subject. Just then Prof. Manju Ray informed that a new laboratory was being constructed at the Indian Association for the Cultivation of Science under her direction and invited me to work there. Prof. Chatterjee gave his consent and I agreed. That day on my way back home, Prof. Chatterjee told me that working with Prof. Ray would open new avenues for me, which would help me in my research. Only the previous year, Prof. Ray had won the prestigious Shanti Swarup Bhatnagar Award, considered to be a coveted science honour in the country. The very next day I went to the Indian Association for the Cultivation of Science to visit Prof. Ray.

On the very first day I told her that I did not come from any scientific background. Apart from a few cases I had treated, I did not have any practical knowledge about the work. For three consecutive days we discussed what to do next and in which direction to move about. At that time Dr. Shelly Bhattacharya was undergoing fellowship under Prof. Ray's guidance. Prof. Ray introduced me to her and asked me to discuss with Dr. Bhattacharya on what I should do next. I thought that I would have to work with Prof. Ray only. However, there did lay a huge knowledge gap among us. I knew that I would be able to do justice with the job I was assigned to do. The work I had done during the '70s also helped me a lot during these days.

The Indian Association for the Cultivation of Science, Kolkata helped me a lot to learn about the basic research methodology of any scientific work. Here I got the idea of difference between normal cell and Cancer cell, and how Cancer cells work in the body of a Cancer patient. Except surgery I gathered many ideas on clinical pathology including reactions of Chemotherapy and Radiotherapy on Cancer patients and other core issues of Cancer-treatment. It is pertinent to mention here that Prof. Indubhusan Chatterjee, an award-winning scientist, and Prof. Manju Ray provided ample cooperation in developing my in depth scientific knowledge. Mrs. Shelly Bhattacharya and others also contributed to a great extent in this research work. In due course, this knowledge helped me a lot while working in the School of Tropical Medicine and the Chittaranjan National Cancer Institute (CNCI) during later years. I will always remain grateful to Dr. Amar Bhaduri, ex-director of the Indian Institute of Chemical Biology. He had once told me that a nation's problems had to be resolved in that country itself with its indigenous resources; otherwise not. I feel privileged to get the opportunity to work with him for some time while I was reporting to Dr. Manju Ray. Every moment I felt inspired when she used to say that she was always ready to guide me in every possible way. But our association did not last long as I moved out from the Indian Association for the Cultivation of Science to the School of Tropical Medicine. I would always relish the fond memory that I began my work with three eminent personalities,

Prof. I. B. Chatterjee, Dr. Manju Ray and Dr. Amar Bhaduri. I will always remember them for everything I have achieved in life and for their genuine guidance by holding my hand in my obscure journey, from darkness to light.



Indian Association for the Cultivation of Science, Kolkata

Until this time my career rested upon the mercy and pity of a few renowned oncologists with whom I shared cordial relations. Gradually I felt the need to study about the very basics of surgery and its scientific dimensions along with its implications. Huge cooperation came from Dr. Chandra, the then chief surgeon of the Chittaranjan National Cancer Institute, Kolkata. He asked me what exactly I wanted to know about surgery. I said that I wanted to know specifically about the role of surgery in detection of Cancer, details of surgical treatment of cancer and palliative treatment. He agreed to help me out, but unfortunately he died shortly. Then I studied the basics of surgery from Dr. Gautam Mukhopadhyay, ex-surgeon Tata Memorial Hospital, Mumbai.

My Days In The School Of Tropical Medicine, Kolkata

In spite of all these activities, until and unless I could get entry into the School of Tropical Medicine and could publish my research papers on behalf of the new NGO formed with the name “The New Resource”, a great uncertainty was looming large over my empirical research-career.

My serious discourse with Dr. Amiyo Hati and Dr. Subir Dutta, and my frantic efforts to procure monkeys in order to facilitate investigation on snake vaccine in that School are still very fresh. In order to get entry there, this endeavour on my part is a matter worthy of mention here. In fact, I was fortunate enough to come in contact with many towering personalities in the School of Tropical Medicine, whose expert guidance enabled me to cross many hurdles with ease and beyond imagination.

As mentioned earlier, I was lucky enough to work with two very reputed specialist doctors, Dr. R. S. Bhakta and Prof. R.N. Brahmachari. They told me that Cancer research would not be possible without participation in any academic institution, because any scientific research needs recognised qualified knowledge in the subject, which comes only

from recognised science institutes. Once, I was returning with Prof. Brahmachari after a check-up of a patient at Garia in the out-skirt of Kolkata, when he told me: ***Things will be easier to learn if you consider yourself as a soldier and not as a scientist. There are so many experiences in your life. If you declare a war, there will be no end to it, and then only, you will be able to learn or use an institution to reach your destination.*** Thereafter, I began to contact the School of Tropical Medicine but there were as many obstructions as one could dare to imagine even. However, I did not march any retreat. One day, I directly met Dr. A.K. Hati. He was eager to know about the purpose of my meeting. I briefed him about my research on Cancer and sought his help. Then he said that it would not be possible to work over there, as it was a government institution. To him I was totally unwelcome. That day I left the place silently, but later I unashamedly again went back to meet him. He received me well but asked me politely as to on what right I intended to work there. I politely replied: ***I have come with a passion for work on Cancer medicine and for that I wanted to reach the School of Tropical Medicine. This School only possesses the scope of modern scientific clinical research and I dream to avail of that scope to take ahead my research on Cancer. I have not come with any job expectation or anything else.*** Prof. Hati repeated: ***Look, minimum qualification to do research work in this type of Govt. institution is the academic degree of MBBS/MD or M. Sc. in Biochemistry or Physiology. Sometimes, exceptional opportunities are provided in foreign countries but no such provisions are available in India. Cancer is a specialized as well as a highly complicated subject. Here, we may have Haematology subject (in the Blood Cancer Unit), but again this is not a Cancer hospital.*** Finally his words were: ***I am busy now, and you may leave.***

I, however, continued to go there. I met Ms. Pritha Dasgupta, a social worker. Dr. Hati also knew her. One day Ms. Pritha Dasgupta, Shri Gautam Saha and myself went to Dr. Hati's house. He welcomed us but refused to join with us in any academic discussion. We returned in despair. Later, Ms. Pritha Dasgupta and Shri Gautam Saha separately met him and Dr. Hati agreed to come to academic discussions with me. He asked me to show a biopsy slide of a particular case. One Bijoy Kumar Hui had been suffering from stomach Cancer and he was admitted in Chittaranjan National Cancer Institute but later he was under my experiment. It was difficult to get his biopsy slide. Smt. Utpala Chatterjee, the then Deputy Director of the Chittaranjan National Cancer Institute, asked me to apply for it and I finally got the slide with the help of Dr. Hati. I could convince the authorities at the School of Tropical Medicine with the slide about the success of the therapy, and Dr. Hiranmoy Mukherjee ultimately agreed to reconsider my matter, though he was not satisfied with the papers only. He wanted me to have practical result shown by experimenting on patients. I was asked to face an advance-bladder-Cancer patient, one Mr. Ganguly, whose rectum, nose and mouth were bleeding. He had ascitis and Cancer spread all over his lung and liver. Dr. Hiranmoy Mukherjee asked me to apply my therapy on this critical patient, and to show the result. Indeed I already had the experience to deal with such kind of patients, but at that moment, situation was different, as I had to face the challenge from the scientific world. However, with the grace of God, I succeeded in getting the patient's condition improved a little within three days with all cooperation from Dr. Subrata Bhattacharya, the then RMO of the SSKM Hospital, Calcutta. There was a problem at the

time of providing the patient with two bottles of blood but somehow it was managed and his condition improved a lot. Finally Dr. Hiranmoy Mukherjee and others were convinced and I was permitted to work on my therapy in the School of Tropical Medicine.

In the meantime they needed some monkeys for testing of a snake vaccine and asked me whether I could provide monkeys to them within a week or so. I accepted the challenge. They admitted that this was a difficult task. We were seriously trying to collect monkeys for many days, but in vain. We even tried to buy monkey from New Market. We went out almost everywhere to search for monkeys and finally we got one at south of North 24 parganas but other monkeys attacked us and the local public vehemently opposed the venture. So we had to give it up. Days passed, we could not collect monkeys. Mr. Pradip Kundu, Mr. Tapan Sen and Mr. Tanmoy Bhounik of our team broached the idea of collecting monkeys from those who used to arrange monkey-shows on streets in exchange of money, and the idea clicked. So, finally we could arrange monkeys from Nilganj, Barasat. People at the Tropical were equally surprised and happy, and thereby I got the scope to study as well as to engage myself in experimental works there. Gradually, I could build up a lasting relationship with many in the School of Tropical Medicine, like Prof. P.S. Chatterjee, Prof. P.K. Kundu, Prof. Partha Banerjee, Dr. P.K. Bhattacharya, Dr. Debasis Basu and others. I was fortunate enough to use the rich library of the School to prepare my papers. I developed contacts with Entomology and Nutrition Department of the Tropical to gather different scientific knowledge. Many patients used to come to meet me at the Tropical. All these things were very much beyond the conventional and administrative sphere of rules and regulations. Shri P. K. Kundu in particular cooperated with me a lot in the field of medicine. Several times he accompanied me to the residences of Cancer patients.



The School of Tropical Medicine, Kolkata

In due course I got the chance to work in the hospitals basically through the auspices of the School of Tropical Medicine. This was the time when I came to know about minute details of Cancer from doctors like Dr. Anup Majumdar, Dr. Jaydeep Biswas, Dr. Abani Chandra, Dr. Utpal Chatterjee, Dr. R.N. Brahmachari, Dr. R. S. Bhakta, and Prof. (Dr.) Subir Datta. After all these developments, when I tried to be close to the Grade-IV staff, they often hesitated to open up and tried to avoid me. Although some patients' condition used to improve and the hospitals used to record such improvement, still it was difficult to collect official documents

from them. Sometimes we had to go to the patients' residences on the pretence of delivering to them food or fluid.

Here I would like to mention one case that I came across. When I joined the School of Tropical Medicine I had to face some resistance from a few doctors as well as the hospital staff. People raised questions as to how someone can enter the prestigious institution like this and start performing experiments without any sound medical background. One day someone even asked me: ***Are you an employee, a doctor or a scientist? What business do you have there? Why do you visit patient's wards and the institution's library?*** The people who used to love and respect me at the School of Tropical Medicine defended me and asked him who was he to ask me that question. After all this, when I came back home from the School of Tropical Medicine, I was not feeling good about the entire episode and did not go back to the School for near about ten days. On the tenth day Dr. Mukherjee called up and informed me that an incident had taken place. He asked me to arrive at the School of Tropical Medicine as early as possible. I hurriedly went there. When I reached there, I found Dr. Mukherjee and two hospital staff, Gurudas and Sukumar, to smilingly welcome me. I enquired as to what had happened. Sukumar replied that after I had left, some people engaged in the Calcutta Medical College while opposing my work on the plea that we were violating the rules of the Institution i.e. of the School of Tropical Medicine, suddenly one of them rushed to the washroom. Unfortunately instead of passing stool the person started bleeding profusely. Colonoscopy was performed. A mass was observed in the rectum along with ulcer. Then from that mass, biopsy was done. By looking at the colonoscopy report the attending physician suspected it to be a malignant growth. After a few days when the biopsy report came out it diagnosed adenocarcinoma and the sonography report suggested two metastasis in the liver. The person was a stage-4-cancer patient. His name is Mr. Bimal Adhikary. Although he was an advanced stage cancer patient, somehow his general condition was not really bad. He was advised to undergo Chemotherapy but he refused. A few units of blood had already been given to the patient by the time I visited him. People at the School of Tropical Medicine advised us not to take up his case, but we took up his case as a matter of principle of humanity and in right earnest. For me the patient is always the priority and by that time I had also learnt that his wife was pregnant. In my attempt to treat and cure him, I applied Psorinum Therapy on him. Gradually the patient's condition improved. The metastasis in the liver disappeared. Later on, the Board of Doctors decided that they would surgically remove the colon. When Prof. (Surgeon) Madan Choudhury performed the surgery, he observed that the mass showed no growth. The patient was an employee of Indian Railways but on leave to do works of the trade union. Since at that time the patient was recovering from cancer and had a family to support, he joined back his old service. He is still alive with active habits and is now one of our proclaimed well-wishers. This was followed by my uninterrupted research work for many years. For nearly 12 years we did not hear from him. Suddenly on 02.05.2016 he visited my facility centre and on the same day also met Prof. Anup Majumdar and Dr. Mukherjee who had come to my centre to visit other cancer patients. All of us were very happy to see him after so many years. We all took some photo with him as well. I asked him whether he had his old medical reports to which he said: *Yes, but not all. A few documents already got lost.*



Anup Majumder, Hiranmay Mukherjee, patient Bimal Adhikari and Asim Chatterjee (left to right)

I am placing those medical documents here. Without his reports the documentation of my journey so far would have remained incomplete. Currently he is a retired person and residing in Rourkela. He had come here to consult me regarding a cancer patient's treatment.

Reports and supporting documents of Bimal Adhikary

E-CG - NAA CXR (10/01/95) NAA

Barium Enema (29/12/94) Structure at the recto-sigmoid junction.

XRay L/S spine (03/01/95) \leftarrow AP \leftarrow Lat : all vertebrae show mild degree of demineralisⁿ.

Colonoscopy (13/01/95) upto junction of sigmoid is descending colon (at 35 cm. from anal verge) An intra-proliferative lesion is seen at that area involving all the quadrants. ~~Endo~~ Colonoscope can't be negotiated beyond this.

Biopsy - (Colonic biopsy consists of 6 bits of tissue).

No overt malignancy is seen. The mucosal bits show adenomatous change (? Polyp) suggest close clinico endoscopic follow up.

Advice on discharge

1. Terramycin (250mg) 1 Cap. 6 hrly x 7d
2. Ref. to Gastroenterology dept of SSKM Hospital on any Mon day / Friday at 9 AM.

Bimal
21/1/95

Regd. No. 914

Carmichael Hospital For Tropical Diseases

School of Tropical Medicine

West Bengal Form No. 788 Calcutta-73

26/12/94

21/01/95

TICKET FOR OUT-DOOR PATIENTS

C.H.T.D.

At the..... Hospital

Date of first visit..... No. in C. P. Register.....

Name.....

Age..... Sex.....

Occupation.....

Presenting P/L for investigation.....

Diagnosis.....

Investigation:-

Date..... Treatment.....

Urine for R/E (28/12/94) N.A.D.

Stool for Ova (29/12/94) N.A.D.

Stool for R/E (28/12/94) N.A.D.

Stool for Occult Blood (29/12/94) Strongly positive

Hemogram (03/01/95) Hb 13.7 gm%

PCV 0.45 L/L TLC 4,900 /cmm

Platelets Adequate N67 L29 M2 E4

PPBS (06/01/95) 100 ESR 19mm in 1st hr

Cholesterol (09/01/95) 262 mg%

Hemogram (10/01/95) Hb 12.2 gm% TLC 6,500/c

N46 L46 NACIP-A 30405-1977-78-2,64,000-2,64,00,000

N46 L46 N3 E7 ESR 08mm in 1st hr

B-Urea (14/01/95) 26 mg%.

(4)

Date

Treatment

S.S.K.M. HOSPITAL
CALCUTTA

Adenocarcinoma Sigmoid Colon

27-12-95

Ward A (Monday)

NAMES LISTED UNDER

PAID IN ADVANCE

DATE

ENLISTMENT NUMBER

62 Shakti 27/12/95

Clinical Tutor Cum R.M.O.

Department of Surgery

and S.S.K.M. Hospital, Calcutta-20

ANESTHETIC CHECK-UP

DAY ROOM No.

Seen

To submit

ticket in

1-2-2006 Shakti 31/12/95

Admission in Curzon

Ward under unit A

(Monday) as G.O. free

62 Shakti 31/12/95

Department of Surgery

S.S.K.M. Hospital, Calcutta-20

Rm. 502/7 Re. 11-

S.S.K.M.

TICKET FOR OPD PATIENTS

No. D 150070 Date of Issue 27-12-95

HOSPITAL

Name Bimal K. Datta

Age 48 yrs Caste Sex

Disease

Date Treatment

Adenocarcinoma Sigmoid Colon

Admission

Admission for operation

(a) Anesthetic check up

(b) S. T. Toxoid 1st dose

(c) S. T. Toxoid 2nd dose

(d) Blood for R.E.

(e) Blood for Sugar, Urea, Creatinine

(f) X-ray chest - P.A. view

(g) S.C.G. in all leads

Dr. M. M. Chowdhury (Reader)
Dept. of General Surgery
I.P.G.M.E. & R.S.S.K.M. Hospital
Calcutta-20

S.S. K. M. Hospital / Raipur

Dr. M. Chowdhury

West Bengal Form No. 817

No. 19831

DISCHARGE CERTIFICATE

Cur 200-5

I hereby certify that BIMAL ADHIKARY 48/M/H C/O.
Ranjit Chatterjee of 24/9, B. P. Ganguly St.
Cal-12 P.S. Bowbazar was under treatment in this

Hospital from 7/8/95 to 4/9/95

Suffering from Adenocarcinoma Sigmoid Colon

SSKM. Hospital

Signature

The

4/9/1995

Designation

PGT

Bl. 961 B positive.

There was an atmosphere of cooperation all through during Dr. Hati's Directorial tenure. After his retirement, reports were published in 'Aajkal' (a Bengali newspaper), criticizing our work as lacking in legal pre-requisites and sanctions. Our working ambience gradually deteriorated and ultimately our project was abandoned. Our regular works at the Tropical was stopped but contacts with many doctors of the School of Tropical Medicine continued. After this incident, I steered forward my Cancer project in persuasion with Tata Memorial Hospital at Mumbai.

It was really a tough job to work on Cancer during those times. Even advanced medicines for simple headache were not easily available. I often found myself surrounded by both friends and foes in the hospital. There were attempts to destroy vital documents of my patients or their biopsy slides. One S. P. Deb Roy was a 62 year old advanced/critical Cancer patient who remained admitted in the Intensive care unit of SSKM Hospital, Kolkata from 21.12.1991 to 13.02.1992. He was admitted for frequently passing black stool for last 6 months and with a complaint of gross reduction of weight, diminished appetite and Bronchospasm. He had suffered previously for Respiratory problems and was diagnosed as a case of COPD. Upper GI Endoscopy showed ulcer in the lesser Cervature of stomach without any evidence of Haemorrhage. During his stay in the Hospital he became grossly emaciated (B/W measured 35 kg.) and Anemic, associated with constant uncontrolled passage of stool, most of the time black in colour, having a neuritic odour, and slight Bronchospasm further associated

with haemoptysis once. In colonoscopy an ulcerated area was found in the sigmoid colon from which biopsy was taken which showed cells of mucous secreting adenocarcinoma of rectum on H/P examination. Blood Transfusion and other supportive treatment were given to the patient during his stay and he was being referred to Chittaranjan Cancer Hospital for Bachitherapy.

I knew a lady attendant in that hospital who informed me that this patient was admitted in the Critical Care Unit of the hospital and the doctors were working very hard on that patient. She also said that the patient was being shifted to Chittaranjan Cancer Hospital and his relatives had already called for the ambulance. I reached the hospital immediately. The lady attendant asked me not to enter the hospital then and there. She called the patient's relatives to meet me outside the hospital. I told them that taking the patient to Chittaranjan Cancer Hospital might not be very fruitful. The patient's relatives also agreed to this fact. I had a discussion with them for around 15 minutes at the end of which I suggested that the patient be admitted to a local nursing home but his relatives decided that the patient should be taken to his home. I went with the patient in the ambulance to his residence at Birati. Since the patient was having some breathing problem, we arranged for oxygen in the patient's residence. I also called for a resident doctor, Dr. Roy, to attend the patient. I stayed there for 48 hours till the patient's condition became stable and then Psorinum Therapy was started since 14.02.1992. His overall condition improved remarkably in the first two months.

Meanwhile, I tried to contact one of the attending doctors of the patient. I knew that the patient was being attended by Dr. A. Konar, Dr. Subrata Bhattacharya, RMO in the SSKM Hospital, and by Prof. Guha Majumdar, a famous gastroenterologist and the then Head of Department of SSKM Hospital. Again the then Director of SSKM Hospital, Dr. D. Sen, a senior chest specialist, was also looking after the case. After many attempts I realized that none of them was interested to meet me. Just then the above-mentioned lady attendant extended a helping hand and convinced Dr. Subrata Bhattacharya to meet me. It was Dr. Subrata Bhattacharya who said that if in 2 months they could not do anything I would also not be able to do anything. When I first met him he asked me how the patient was. Where was he? I replied that the patient was at his own house. Then he asked whether the patient was alive or not. I replied yes. I invited him to visit the patient at his house for once. He agreed to it. The next day in the early morning I picked him up from his house at Dum Dum Park and we went to Birati. I also requested Dr. Roy to be present there at that time. Dr. Bhattacharya was totally surprised and at the same time very happy to see the patient back on his feet, and simply remarked that it was a miracle. We then started visiting the patient too often. The patient was also gradually recovering.

After considerable improvement the patient reported to the SSKM Hospital on 20.04.1992. Dr. A. Konar, Assistant Professor of I.P.G.M.E.R., Kolkata examined him in the outdoor for repeating Sigmoidoscopy and Rectal biopsy. Accordingly Sigmoidoscopy and Rectal biopsy were done on 28.04.1992. Sigmoidoscopy passed upto 12 cm. and no internal mass was found and an 8 cm small ulcerated area was found. Biopsy report of the section showed histology of rectal mucous with features of chronic non-specific inflammation. Mr. S. P. Deb Roy was certified to have been fully cured through Psorinum therapy. Dr. A. Konar

acknowledged the success of the therapy and gave me a written acknowledgement letter to that effect on the hospital's letter-head. This was also not easy for both Dr. A. Konar and Dr. Bhattacharya and I thankfully appreciate them for this. S. P. Deb Roy's case emerged as a successful case and as a miracle tale.

Mr. S. P. Deb Roy's documents were tried to be destroyed in the SSKM Hospital. Anticipating this conspiracy, I somehow with the patient's help managed to collect all his documents including biopsy slides. This led to a big commotion. Needless to say that I had to stay in this patient's house for three months to apply my therapy. Dr. Subrata Bhattacharya and Dr. Roy cooperated a lot with me in this regard. I remain extremely grateful to them as well as to the lady attendant who helped me a lot in this case.

Here I would like to provide all the photocopies of those documents that I have as a valid proof, regarding Mr. S. P. Deb Roy's treatment and his recovery.

Mr. S. P. Deb Roy was leading a normal life for 7 – 8 years and later he died of old-age disease. But before his death, he handed over all his medical reports and documents to me thinking that those would be of any help to me in future.

Reports and supporting documents of SP Deb Roy

SI No. 115

Month Dec

Reg. No. 23426

INTENSIVE THERAPY UNIT

CLINICAL RECORDS

Name Shibaband Debroy

Age 70

Sex Male

Date of Admission 2/12/91

Date of Death

Date of Discharge 14/2/92

DEPARTMENT OF MEDICINE

S. S. K. M. HOSPITAL

1991-1992

FOLLOW UP CARD		
Intensive Therapy Unit Department of Medicine S. S. K. M. HOSPITAL		
Sl. No. 125	Reg. No. 23424	
Name Shibaprasad Debray 66 yr H.M.	Age 66 yr	
Address % Anurag Sub Ray 282 Nisachal, Patnagar, Cal-51	Sex M	
Date of Admission 21.12.91	IT.U. 27.12.91	Date of Discharge 14.2.92
Diagnosis (i) Ca Rectum (ii) Metastasis due to small nodule on liver edge. Stay in IT.U. (iii) COPD - hydro-pneumothorax.		Consultant Dr. A. Konar
Clinical Note Treated conservatively		
Treatment		
Condition on Discharge Satisfactory		

K. S. 14/2/92

Investigations:		1.1.92		10.2.92	
Na ⁺ 26/10	141 meq/l	141 meq/l			
K ⁺ 5.2	4.5 "	4.5 "		54 mg/l	
Sugar 72 mg/l		45 mg/l		36 "	
Urea 120 mg/l		1 mg/l		0.6 "	
Cr 2 mg/l					
L.F.T. - bilirubin 0.7 mg/l	0.6 mg/l				
T.P.A. 5 %	5.9 %				
Alb 2.8 g/l	3.6 %				
Celob 2.2 %	2.9 %				
Alk. Phos 75					
SGOT 300 U/L	69 U/L				
SGPT 100 U/L	71 U/L				
	SGT-1310/L				
4.1.92		23.1.92			
B.T. Trophozoite present in peripheral blood film		Biopsy from rectal ulcer -			
10.1.91 (consecutive 3 days)		Shows - histology of mucous secreting adenocarcinoma with ulceration			
R.F.B - RF in Sputum					
22.1.92					
ESR - 1st hr - 45 mm					
2nd hr - 80 mm					

INTENSIVE THERAPY UNIT
DEPARTMENT OF MEDICINE
Institute of Post Graduate Medical Education & Research
S. S. K. M. Hospital, Calcutta-700020

CLINICAL RECORDS

Serial No. : 125 Cubicle No. : 3 Reg. No. : 28424

Patient's Name Shiba Prasad Debroy Age 66 Sex M

Occupation Retired Address C/o Anand Debroy, 282 Nibhat
Burahi, Calcutta-51, P.S. - Anandpur.

Admitted in hospital on 21/12/91 (Word NEW) Under Dr. A. K. Das

Admitted in I.T.U. on 27.12.91 Initial Diagnosis Struma hyperthyroid
Malaise.

Reason for transfer to I.T.U. —

Date of discharge/death 12/1/92 Stay in I.T.U. 1 days

Final Diagnosis —

Presenting symptoms with duration —

Unconsciousness —	Giddiness —
Dyspnoea —	Nausea —
Palpitation —	Vomiting —
Syncope —	Anuria/Oliguria —
Convulsion —	Oedema —
Precordial pain —	Others —
Sweating —	

Past illness —	Bruchitis —
Diabetes —	Asthma —
Hypertension —	Rheumatic Fever —
Nephritis —	
Drugs —	
Trauma —	
Others —	

Family history :

Personal history (Obstetric history in case of female) :

Brief therapeutic measures already taken :

Examination :

I. General Survey

State of consciousness

Anaemia

Jaundice

Pulse

Respiration

Temp.

B. P.

Neck vessels

Conscious/drowsy/stupor/coma.

Cyanosis

Clubbing

Oedema

Nutrition

Skin

Other

Neck glands

II. C. V. System J. V. P. —

Pulse

Rate

Rhythm

Volume

Character

Other peripheral pulse

Apex

Position

Character

Sounds

M.A.

A.A.

P.A.

Heart sounds

and sound

I.V.P.

II.V.I.

(0)

Follow Up

	Date : & Time	Findings	Remarks
28/12	At. Conscious H/L - n. n. s. Cough present. Cline / n. s. Splen. / n. s. Kalamia in small amount present.	96 $\frac{110}{70}$ 24/12 11/12	
28/12 (6)	No nasal mucus. H/L - ✓ Upper abd. back infection L/R / n. s. ABD. Soft Sp. s. t.	100 $\frac{130}{80}$	Upper G.I. Endoscopy done. No ex. cure present. No evidence of SRA.
			Ph. x/p H/L / 70 D and urea. Estimate on 80/12/01 Send stool for R. Exam
28/12	No nasal mucus. H/L - ✓ ABD. Soft.	90 $\frac{110}{70}$	30/12 12-10 pm. Blood transfusion - one bottle. 5-10/10 G. C. + R. 150 drops / min.
31/12 11	No nasal mucus. H/L - ✓ ABD. Soft.	92 $\frac{110}{70}$	21/12 H. made no more blood transfusion - Acquisition for E.C.G. done.
31/12 18	No nasal mucus. Ectopic breath + Abul - soft No brucellosis	96 $\frac{125}{70}$	

	Date : & Time		Findings		Remarks
1/1/92 m	Pt. Conscious. / on med. H. 1100 Temp. 100 No hge. or flow	92 $\frac{120}{80}$		Blood transfusion started at 1 pm on 1.1.92 Bottle no. - D - 11353 O + ve	
2/1 K	Conscious. Small amount melaena 3 times since last night. H/L - 1100	86 $\frac{130}{80}$	2450 950	In phs A. 11 - 1000 2/1 Blood transfusion started on 2.1.92 at 12.40 Bottle 11352 O + ve	
3/1 (m)	c/o Fever no melaena. H/L - ✓ 1000	78 $\frac{120}{70}$	1500 700	Blood for TC done (5R sent (normal).	
4.1 H	Pt. Conscious. Small melaena last night. ang. ulcer stomatitis adema left foot	88 $\frac{140}{80}$	1400 800		
4.1 (5)	conscious. Fever - 104.0°. no blood passed y/h. H/L ✓ 2	78 $\frac{130}{80}$		Blood for m.p. sent.	
5/1 m	Temp. 100°	86 $\frac{140}{80}$			
6/1 (m)	Pt. Acting normally. Stomach contents	88 $\frac{110}{70}$		1. Blood reg. done. 2. Blood for Hb / TC 100 mm sugar.	
7/1 m	Mela.	11 110	11 110		

11/10	Conscious. Responsive	72	$\frac{110}{70}$		Blind for vision
11/10	Cooperative				Went, dead, 11/10
	melena +				for fine diet
8/1	melena +	72	$\frac{110}{70}$		
9/1	at conscious, restless,				Bl transfusion
	o/o throat & trachea;				Butte No. 8482
	small amount of melen				Collected on - 3/1/92
	frequently. H/P. N. & D.	116	$\frac{178}{146}$	500	Exp. on - 18/1/92
	alid. appears slightly				O + ve. hemolysis
	rigid. Malena				3 phosporan 1st
	appears increased:				9th Sol
	temp - 100.4°F				to donor 1 amp 9th
					serum half of
					the bottle has been
					transfused
					Surform no. Sol.
					Wt - 45
					Cr - 1.0
10/1	C/O Haemofitosis	96	$\frac{160}{80}$	500	1) Blood for H&G, T.C. ne
(m)	- in the morning.				of R/T/Enzyme
	melena - continues.				- 31st.
	H&A - V				2) Blood transfusion - 1 bottle
	of crepts (-) Lids				given.
	(2) apical.				(NO. 8547) 600 cc
	600 + lower				
	2000.				3) Sh. & Ray C host, (if any)
11/1	Phlebotomy performed	96	$\frac{130}{90}$	500	Barium Ba-enema
	with muscle guard all				to be done.
	over abdomen, 1 P.S.				
	Normal, lungs full				Pl. send sputum
	of crepts & Rhonchi				for AFBX 6 days.
	temp - 100.8°F				
11/1	melena - continues	88	$\frac{130}{80}$		
(E)	Temp - 100.2.				
	H&A - V				

Wagon
Hill

Pt. is real bad & c/o
discomfort at uterine
region.

8486 13/1
3/1/90 (m)
17/1/90
on as usual
you last 14/1
m
1/2 cup of
self of
1/2 been
ne. Sel. 14/1
E

Temp 99.0 F
no hemophysis
melena. 8/255
NA/L
S -
Temp 98.5

Condens
lungs. No bronchopneum

15/1
M. melena +

Pt. conscious, oriented.
Epigastric pain severe
subcostal. Epigastric
point-tender.
no melena since last
night. H/A. N/A
melena - purulent.

HEALTHY DE
me
H. sm. 1 bottle
given.
7 6/20/90

Host, (P. A. V.)
Ba-enema
1

burum 17/1
& 6 days. (m)

no melena.
NA/L

18/1
(F) melena 1

19/1
m loose melena
No melena
1/2 cup of
1/2 cup of

100 130
(R) 80 140 140

72 130
80 140 140

92 130
80 140 140

76 140
80 140 140

82 140
84 140 140
92 140
84 140 140

06 140
(R) 20 200 600

90 130
75 75

72 130
75 75

B
Brow to be sent
for culture etc.
(15/1/90)

Endo. Joy on 15/1

- Ba. near failure
it's enough on 16/1
partly still takes the
pt. 9 Am
only prescription
in pt will not take
amblyed tomorrow

Sputum -
AFB - Not found
J. Bil. sm. 6 mg
J. pot. 5.9 2.7/1

ACT - 69
AST - 71
2 GT - 13.5 u/L

Alcohol sent for
picluri -

Blood measurement done
on 25/1/90
15/1
Ht-106.5, Wt-5000
HE, E, L30, M2
Platelets - 224

	Loose melena + no mekna Temp - normal	72 $\frac{130}{90}$	1302	730		+
41	Continued blood no melena	76 $\frac{130}{90}$				E
41/1	Loose melena Temp - normal	72 $\frac{130}{90}$				(M)
43/1	pt. conscious no mekna			150		
24/1	no melena / vom loose stool - 3 times	96 $\frac{130}{90}$	150	150		E
	St. ceph (+) St. m. zone + lower	(15)				1.2 (E)
15/1	pt. conscious H/L - N.A.D. abd - soft	110 $\frac{120}{80}$	1400	150		2/2 E
25/1	no melena stool - frequent H/L - V	88 $\frac{150}{90}$				2.2 E
26/1	stool - frequent H/L - V	88 $\frac{150}{90}$				2.2 E
27/1	no melena H/L - V	80 $\frac{130}{80}$				
G	Conscious No vomiting Ankle injury right leg displaced	90 $\frac{120}{80}$				
28/1	Conc. Temp - normal	72 $\frac{120}{80}$				4/2 E

Blood transfusions
given - mekna.

28/1
Pt. has been seen by Prof.
D. Sen. Adv. chest X-ray
today & show him the plate.
X-ray unit main block has
refused to do it today.
Requisition done.

29.10.12

Stool R.E - N.A.D.
C.R. - pleural effusion increased.

20/1 Conscious, alert, self.
loose stool persisting

96 130
86 185

E Conscious.
Very much worried about
constant defecation &
fluidity of stool.
Stool has a NECROTIC
odour.

92 120
90 70

21/1 (M) Conscious. Worried.

Ht/V ✓
no fever
no stool
since morning

96 120
80 1750

CXR is seen by
Prof. D. Sen.
As Dr. Kumar is pl.
see it and instruct the
next work up.

E Conscious. Very much worried
about defecation report.

90 140
90 70

1.2/2 Conscious, Ht/V. N.A.D

96 120
86 155

1.2 (E) Conscious. Worried.

Ht/V ✓
ABD

112 120
(R) 80

2/2 m. Same.

100 120
80 175

E Pt. Conscious. Ht/V. N.A.D

96 130
90 90

2.2/E Continuous defecation +
incontinence which is the
most important of the
features.

90 140
80 17

G.C. L.S.
Morally very much depressed.
Inherent fears to undergo
any surgical procedure.

PATIENT KNOWS THAT THE
BIOPSY REPORT DID NOT ARRIVE
TILL DATE

(— about which he asks
very frequently.)

Same.

92 140
80 170

NO 657/92

SECTION SHOWS
HISTOLOGY OF
MUCOUS SECRETING
ADENOCARCINOMA
WITH ULCERATION.

Dr. D. B. Banerjee
Professor
Dept. of Pathology
IPMER
Dt: 3.2.92

1/2	Conscious. H/L NAD	74	130	20
1/2	MO SACC.	80	140	20
1/2	H/L	80	140	20
E	Same.	90	140	20
9/2	Pt conscious H/L NAD	96	130	80
10/2	abd. Soft.	96	130	80
8/2	Pt. (conscious)	88	150	80
10/2	H/L - L. O. R. (t) with Aus. field.	88	150	80
7/2	Same.	92	140	50
9/2	Pt. conscious. Oriented. Mentally peaceful. No diarrhoea/melena.	88	130	80
10/2	H/L - L. O. R. (t) with Aus. field.	88	130	80
11/2	Same. Junc. (t) abnorm. No Melena. SOGm.	80	130	20
12/2	Same.	90	130	20
E	Conscious. Responsive. Cooperative. Oriented.	94	140	70
14/2	Same. No melena.	92	130	20
15/2	Conscious. Alert. Slightly better. No Melena. Same.	100	130	80
16/2	Pt. conscious. H/L - NAD	106	120	20

1680
800

9/2
Pl. send Blood pr. -
TC DC Hb%. ESR
to Room 16
(H/T/Enzyme, Na⁺, x⁺)
to G.E. Lab
Urea, creatinine to
Bio. chem.
on 10/2 (morning)

Urea - 56mg%
Creatinine - 36mg%
Hb - 10.9 G%
N L E M

Not sent today
Please send a.m.

Platlet -

12/2
Hb - 10.9 G%, TC - 6100
Hb, E, L, 30, H, 2
Platlet - 200,000

West Bengal Form No. 709
Ticket for Out-Door Patients

Hospital
Date of first visit: 20/4/92 No. in O.P. Register
Name: Siba Padm Deb Roy
Age: 65 Case: H Sex: M
Disease:

Date	Treatment
20/4/92	Had been admitted to ward for fecal incontinence + rectal ca → due to suppurative gash in rectum on H ₂ O ₂ + COLD + pneumothorax No Rx
	75 An ⁺ BW 35 Kg Chest: Emphysematous Heart: ✓ Abd: Soft NAD.



A.R.

- ① Sigmoidoscopy + Rectal biopsy
- ② Rubeplex = 2 tap b.p.

To come for sigmoidoscopy
on 28.4.92 (Wednesday)
at 11 a.m. to C.T. Block
after taking Proctoelium
catheter at 7.30 a.m.

[Signature]

S. S. K. M. Hospital, Calcutta

Ward No. Bed No.
Name Siba Pada Deb Roy 65/H/M

Sigmoidoscopy done on 28/1/41

P/R + proctoscopy - no internal piles
or mass felt.

Scope passed upto 12 cm. At
8 cm. from anal verge there is
a small ulcerated area &
started to bleed on touch. Mucosa
below and beyond that normal.
Spec. taken from ulcerated area.

Date. H. S./H. P.

Reg. No.

Instructions to patients/patient's guardian.

1. Please scratch and sign the label of medicine phials before delivery.
2. Please take unused medicine back when leaving hospital against receipt.

P. T. C.

West Bengal Form No. 507

S. S. K. M. Hospital
Clinical Report Form

No. 70/2001 3657 Bed No. 401 Ward 654.5 sec. 1.6.66

Name P. D. Debroy

Clinical Diagnosis Rectal Mucosa 92 HP 1200

Materials for Examination C/O fecal incontinence + melaena : 2 c.c.

Specimen how taken gic + NAD 89/107 show converted by mucous mucus

Nature of Examination required 2 one hr of intonation

Short History Signature of Referring Officer

Date 22/4/1992

REPORT OF EXAMINATION

Pathological Record No.

Date. 19

Clinical Pathologist

3051/02

4.5.02

Section shows histology of rectal mucosa
with chronic nonspecific inflammation.

Prof. J. C. Das
M.D. (M.B.)
Prof. J. C. Das
F.S.M. (M.B.)
Calcutta 700 004

DR. RABINDRANATH BRAHMACHARI
M.B. (M.B.)

Ex. Assistant Surgeon
Professor and Head of the Department, Radiology (Therapeutics)
Institute of Post Graduate Medical Education and Research
Calcutta

A. S. 74, SALT LAKE CITY
CALCUTTA-700 064
34-8550

Old case of Bronchial asthma

It was on Steroids for a long time
It was smaller in S.K. & T.D.
On 21.12.51 with malena.
He was treated conservatively
with Blood transfusion and other drugs
Upper G.I. endoscopy - ulcer in the
lesser curvature
Lower G.I. endoscopy done on last
wk of Jan '52 - mucous secretions
adhes carcinoma.

It at present

G.C. fair - B.P. 115/80 mm Hg
Synthesis done again on 28.4.52
No malena at present
abd - soft - Liver spleen not
No tenderness.

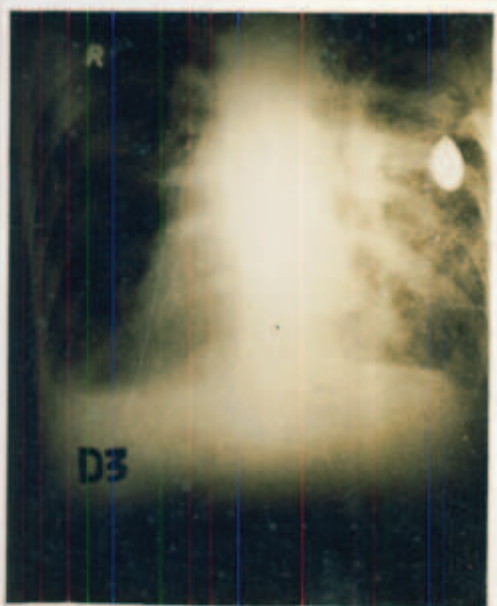
Shiva prasad Deb Roy
66 H. W.

Bromin Symp - 115 3 times a day.

Quinaplex 115 B.D.

Rabindranath
5/5/52

To be review after H.P. report.
C.B.





SITTING - LEFT - S.P. DEV ROY
 RIGHT - DR. ASIM KUMAR
 CHATTERJEE

I would like to provide here a paper that was published under the School of Tropical Medicine's Medical bulletin –

NON-CONVENTIONAL TREATMENT OF CARCINOMA: STUDY OF 52 CASES

Chatterjee A.K. The New Ressource, Calcutta.

**Dutta SK, Kundu PK, Chatterjee PS, Mukherjee H, Bhattacharya J
and Hati AK** School Of Tropical Medicine, Calcutta

Bhakta RS Calcutta National Medical College Hospital, Calcutta

Brahmmachari RN SSKM Hospital, Calcutta

Mukherjee BP University College of Medicine, Calcutta.

Summary

Objective evidence of improvement of previously moribund documented patients of malignancy by use of non-conventional agents is presented, although the chemical nature, pharmacodynamics and pharmacokinetics of these “agents” remain to be elucidated. Diagnosis and periodic evaluation (including imaging and histopathology) have been done by reputed oncologists, pathologists and oncotherapeutists. The present series is a compilation from their available health records.

Introduction

A number of papers presented in the recent past in different scientific forums have claimed variable success in the treatment of ‘cancer’ by use of a non-conventional agent “Psorinum” sometimes in combination with other agents 1-7. While the chemical nature of the “agents” remain unclear, it was thought logical for the cause of science, to document in brief the nature of the objective (not just subjective) response in these patients by use of non-conventional “agents” used in conjunction with usual allopathic treatment modalities. The documentation of this response ought to act as an impetus for research into the mechanism of action of the non-conventional “agents”, rather than to disbelieve the responses observed.

Materials & Methods

The types of cases

52 Cases suffering from carcinoma of different organs in stage III/IV were studied. This included carcinoma of stomach (8), pancreas (4), lung (12), large gut (16), biliary tract including gall-bladder (6) and oral cavity (6). The original investigation and treatment of the cases before use of non-conventional agent 52 cases were diagnosed and staged by routine and

special diagnostic techniques including skiagraphy, barium meal with follow-through and enema, fibreoptic endoscopy, ultrasonography and computed tomography. The confirmation was done by histopathology. Initially after detection of carcinoma, each case irrespective of clinical stage was evaluated by Oncological specialists and given treatment by surgery, radiotherapy and chemotherapy, either alone or in combination, according to the merit of the case. All the cases after receiving modern modalities of therapy were followed up accordingly. After initiation of therapy, some patients refused or could not afford or could not tolerate therapy. Some patients developed unresponsiveness to the drugs used, with progressive growth with or without metastasis. Cancer was detected in some patients in advanced stage on first appearance where conventional therapy was not suggested considering poorer prognosis and treatment outcome.

Selection of cases for non-conventional therapy

The criterion for selection was cancer of stage III or IV and performance status (ECOG) of 3 or 4. All 52 cases voluntarily subjected themselves to this open clinical trial.

Non-conventional therapy

Apart from Psorinum and the other “agents” already mentioned, supportive therapy was allowed. This included therapy for infections, for bleeding, for macro and micronutrient deficiencies by allopathic medicines, fluid and blood transfusion as required. All these cases were followed up daily, later on weekly and critically evaluated from time to time.

Assessment of outcome

Tumour size shrinkage was demonstrated by physical examination, skiagraphy, sonography or radionuclide scanning as applicable. CT Scan where possible was preferred for accurate evaluation of tumour size. A partial response (PR) was defined as a 50% or greater reduction in the original tumour mass. A complete response (CR) referred to the complete disappearance of detectable tumour microscopically. Cure denoted complete microscopic disappearance of tumour at the original site. Progression was defined as an increase of more than 25% in the size of the tumour or the appearance of any new lesion. Tumour, markers were detected where applicable and a significant decrease in the quantity of tumour product reflected regression of tumour. The general well-being and performance status were reassessed from time to time. Downgrading of score referred to favorable outcome. The absence of malignant cells in the primary site and absence of metastasis with marked improvement in general well-being, achieving normal activity and survival for a period not less than 3 years after starting therapy is regarded as cure in the present study.

Results

At the start of therapy, all these cases on critical assessment were staged as III or IV having performance status ECOG scoring 3 or 4 and tumour markers were detected in significant quantity in selected cases. After initiation of present therapy, during first 6 months all the cases showed marked remission of presenting symptoms, gastrointestinal symptoms

and hepatobiliary symptoms along with improvement in liver functions. Haematologic response was also quite satisfactory. Significant improvement in ECOG scoring system from 4 or 3 to 2 or 1 was found. Within first year, reduction of tumour size was less than 50% of the original size with an average of 30% shrinkage in size indicating short of partial response. During subsequent follow-up next 6th months, 20 cases (38.46%) died either from infection, bleeding episodes or organ failure. Among remaining cases (61.54%), 22 cases (42.34%) showed further improvement in performance status defined by ECOG scoring and partial response in reduction in tumour growth whereas other 10 (19.20%), cases showed insignificant reduction in size of tumour with waxing and waning of clinical features indicating fluctuating performance status. Subsequent follow-up showed survival of one case of bronchogenic carcinoma for more than 2 years with a partial response in relation to regression of tumour as well as ECOG scoring of 1 in both the cases. Survivals of 2 cases of carcinoma of stomach were about 30 months. One case survived 23 months despite little regression of growth. On the other hand performance status in this patient improved significantly from 4 to 1. The other patient survived 37 months with an improvement in general well-being as well partial response. The most interesting finding in the present study refers to one patient suffering from gastric carcinoma who was living 4 year achieving performance status 0 from 3. In relation to reduction of tumour size, the response was complete regression. No malignant cells were detected histopathologically from primary site. Among cases suffering from colorectal carcinoma, though the mean survived 3 years achieving partial response and significant improvement in performance status. Very encouraging result was seen in another case. This patient achieved complete response in relation to tumour with absence of malignant cells as detected by microscopy from biopsy specimens taken from multiple sites from the area involved. Among 4 cases of pancreatic carcinoma, the prognosis was poor in the present study excepting that 2 cases survived on an average 17.7 months.

Discussion

Survival in stage III or IV cases of bronchogenic, gastric and pancreatic cancer is very low despite modern therapies with major side effects. There is encouraging result after treatment with nonconventional agents with least adverse effects. Though this study is a preliminary observation of only a limited number of cases (52 cases) we are reporting these cases because of the highly encouraging results that have been obtained so far.

Moreover, palliative allopathic medicines like pain-killers, vitamins and other nutritive medicines and electrolytes, and antibiotics to control secondary bacterial infections can be safely administered along with all the above mentioned medicines, without in any way hampering their effectiveness. It is also worth mentioning here that in 2 cases of which one was diabetic, insulin was administered and in another, who had hypertension and ischaemic heart disease the patient was treated by nifedepine and isosorbide and even then the non-conventional “agents” were effective. Definitive therapeutic benefit will have to be confirmed in much larger clinical evaluations of all stages of malignancy. Ongoing study is directed

towards complete evaluation of pharmacokinetics and pharmacodynamics of the “agents”. One must remember that the patients in this series were not referred from this institution for non-conventional therapy. Neither do the authors of this article advocate such non-conventional therapy in lieu of conventional allopathic treatment. The purpose of this communication is to document objective response of malignancies to non-conventional therapy, much of which were evident from the health records of some of the patients who subsequently attended the School of Tropical Medicine for some ailment.

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(Vol.43, No1 -4, 1995 Bulletin of the Calcutta School of Tropical Medicine)

My Venture To The Tata Memorial Hospital, Mumbai

My association with Tata Memorial Hospital at Mumbai is full of more dramatic incidents. Having been debarred from entering the School of Tropical Medicine, Kolkata, I ventured to the Tata Memorial Hospital, Mumbai, with a faint hope of getting destitute terminal patients on whom my research or experiment with Psorinum therapy could be done. With that hope I used to loiter in the corridors of that Hospital and move from one floor to another at random. The security staff had observed me for three consecutive days and then finally intercepted. They asked me about the reason for my movements without any specified purpose and took me to the Chief Superintendent of the Hospital. When I disclosed my identity and stated clearly that I had no bad intention and simply wanted to study how the Cancer treatment was being conducted in such a renowned hospital, they burst into laughter assuming that I might have some mental derailment. They rudely asked me to return home peacefully and not to appear in this area again. I had to leave the premises immediately but after a few days I returned to that place in disguise or make-up with a hope to get in touch with some destitute patients.



Tata Memorial Hospital, Mumbai

This time fortunately I was successful and found a person with fragile health climbing down the stairs with a gloomy face. Anticipating him to be a terminal patient, when I inquired of him, he said his name as one Gautam Pal, coming from Kharagpur, West Bengal and suffering from Cancer. After triple bypass surgery, the Hospital authority discharged him with the remarks that his condition was so deplorable that nothing further could be done for his recovery. He was under the supervision of Dr. R. K. Deshpande, Dr. Swaroop and Dr. Shinde. They checked his health on 5/10/1993 and diagnosed him with Colangio Carcinoma at the head of the pancreas. He returned home and by 10/10/ 1993 he was affected by Hepatitis B, ascitis and infusion in right lung. He agreed to have a last try with our Psorinum therapy and underwent supportive palliative treatment under Dr. R. N. Brahmachari at Calcutta (now, Kolkata). There was no Radiotherapy or Chemotherapy at that time. The patient gradually showed improvements under Psorinum therapy for near about four months and on 5/2/ 1994 Dr. Brahmachari checked the patient again and found him quite stable. On 10/03/1994 Ultrasonography showed normalcy in liver, pancreas and there were no ascitis. I took the

patient to have a re-examination of his present physical condition by the doctors at Tata Cancer Hospital and to have their valuable opinion. I remained aloof in a hotel nearby. The striking improvement of the condition of the patient drew attention of all the doctors. After repeated enquiry, the patient divulged my name and whereabouts. The hospital authority met me hurriedly to know how such spectacular improvement was possible. They also placed some stringent questions and caustic remarks as to whether I had observed international protocol while carrying out the treatment. I made a candid statement that I had tried to save the life of the patient with his full consent and I was simply happy that his life could be saved. They put so many queries and I tried to satisfy them coolheadedly. Thereafter I got in touch with Dr. Dipankar Dasgupta and Dr. Gautam Mukhopadhyay of that hospital, who patiently listened on my invented therapy and my ultimate goal. In course of time they taught me in details the critical Cancer management. Now it happens that terminal stage patients of Tata Memorial Hospital often rush to me at Kolkata to get a last chance to save their lives.

Reports and supporting documents of Gautam Pal

TATA MEMORIAL HOSPITAL

CYTOLOGY REPORT

Tata Memorial Hospital
Dr. E. Borges Mang.
Patel, Bombay 400 012
Tel. 412 5419

CYTOLOGICAL EXAMINATION FOR MALIGNANCY / HORMONAL EVALUATION

Patient's Name

Mr Gautam Pal

Date of collection

13/4/93

Case No.

Doctor's Name

Dr Swaroop

Source of Specimen

Bile

Cyto No.

CYTOLOGICAL INTERPRETATION:

Bile:

A number of flat sheets of ductal cells seen.

A few clusters of cells showing some anisonucleosis,

small nucleoli and hyperchromasia noted.

Adenocarcinoma cells seen.

Date of Report :

19/4/93

Signature :

CYTOLOGY

TATA MEMORIAL HOSPITAL

ULTRA SONOGRAPHY

Rs. 300

Name Gautam Patel OUT PATIENT { Private ☐ Stretcher ☐
 Age 14 Sex M Income Rs. Clinic ☐ Ambulatory ☐
 Requisition Date 6/9/93 IN PATIENT: Wd.
 Amount Charged Rs. 119/26
 T. M. H. Case No. BF 5352 Outside Request No.
 Receipt No. Referred by Dr.
 Referred by (unit Chief) Dr. Chakur Private ☐ Hosp. ☐
 Relevant Clinical Data Ca Head Pancreas No. of 10x6" Films.
By-pass done
 H/O. Previous Treatment
 Examination Requested USG abdomen Anatomical Site
 Appointment on 7.4.93 at 10 am a.m./p.m.
 Reported by Dr. Date: 4th fastig.

BF : 5352 7.9.93 (Reported by Dr. Chakur) (1)

USG OF UPPER ABDOMEN : Liver appears normal in size, shape and echotexture. Intrahepatic biliary radicles and CBD appears normal in diameter but show evidence of pneumobilia. Spleen and both kidneys appear normal. Pancreas appears bulky. Pancreatic duct is dilated and measures 4.2mm in diameter. An anechoic well defined cyst is seen in the tail of the pancreas. It measures 3.5 X 5 X 4.5cms in size. No evidence of any ascites or paraortic lymphadenopathy is seen.

IMPRESSION : USG features show evidence of pneumobilia, with bulky pancreas with dilated pancreatic duct with Pseudo pancreatic cyst in the tail of pancreas.

23.9.93

DR. R. K. DESHPANDE M.S., F.I.C.S., D.H.A.
SURGICAL ONCOLOGIST
T. V. MEMORIAL HOSPITAL
FAREL: BOMBAY 400 012

26
Tel: 4146750-55 (HOSP)
Tlx: 011 73649 TMC IN
Fax: 022 4146937

The Pat is a known patient of carcinoma of
oesophagus who has undergone (radiotherapy &
chemotherapy) + gastrectomy 2 months back

He was recommended here for surgery (total gastrectomy)

However on admission he was found to be having
infective hepatitis B with jaundice and hence
surgery was deferred

He is advised only supportive treatment in
the form of general tonics, B complex etc. He
is advised to have CBC, SALT, ALP, SGOT, SGPT,
S. Bilirubin & C.T. abel after 3 months & send report
here for assessment

23/9/93

R. K. Deshpande (Dr. R. K. Deshpande)

Res: A-22, Shree Gajanan Society, Lt. Dilip Gupte Road, Mahim, Bombay 400 016, Tel: 454187

F. R. N. BRAHMACHARI
DMRT, MD

Consultant Oncologist
Senior Head of the Department of Radiation Oncology
Institute of Post Graduate Medical Education and Research,
S. S. K. M. Hospital, Calcutta.

case of Ca. Colon bile duct
resection May 1983.

supple by-pass (Rugay)

He Ag B -

No lump felt in abd.

✓ 1st.

stomach - Nil

0 -

Free since 1986

Mr. G. Paul

42 Hm.

Adm.

Zentec - 150mg 1 tab at night.

Gellusil - 1 tab B.D.

Pankreatoflat 1 tab B.D. along
with food.

Abrolam, 25mg 1 tab B.D.

Ref to Physical for Control of
Blood Sugar

Dr. Gautam Chatterbadi

30A Nandan Road

(Opp. Police Hospital)

(48-3154)

Gautam Chatterbadi
5/2/94



POLYCLINIC & LABORATORY
279, Kendua Main Road, Garia
Calcutta-700 084, Phone : 72-6064

Ultrasound Scanning Report

Name of Patient.....Mr. Goutam Paul
Age.....42.....Sex.....Male
Refd. by Dr.....Asim Kumar Chatterjee, D.M.S. (Cal), Dip. B.M.
Part examined.....USG of Upper Abdomen
Date.....10.3.'94.....

Liver: Echotexture appear normal with echogenic opacity within billiary channels due to pneumobilia.

Gall Bladder: is not identified.

C.B.D.: .75 cm.

Portal Vein: 1.10 cm.

Pancreas: Echotexture appear heterogenous. Pancreatic duct is about 1 cm. wide. No calcification is seen within the pancreas. No obvious S.O.L. is seen within the pancreas.

Spleen: Shows normal size and echopattern of parenchyma.

Kidneys: Show normal size and echopattern of parenchyma. There is normal cortico-medullary differentiation. No calculus is shown.

Ureters: Not dilated.

No ascites is seen.

No pre/para aortic lymph node is seen.

Impression: Chronic Pancreatitis.
Other investigation suggested.

Jk.
Dr. G. K. Mondal,
D.M.R.D., M.D.

TATA MEMORIAL HOSPITAL

REQUISITION FORM FOR FINE NEEDLE ASPIRATION CYTOLOGY

CASE NO. BF 5352 UNIT : Indraprastha DATE 13/6/94
PATH. NO. _____

NAME : Gautam Paul

AGE : _____

CLINICAL DATA :

Pneumatic Mass



F-NAC

SITE OF FNAC :

NATURE OF MATERIAL : (MACROSCOPIC APPEARANCE)

NO. OF SLIDE : _____

PREVIOUS PATH. NO. : _____

FNAC REPORT : _____

DR. _____

appointed the Secretary of the newly formed NGO, namely, the 'New Resource', and Mr. Tapan Sen was chosen as the Chairperson.

The New Resource also played an important role in many respects like searching for new Cancer patients, settling them down, providing facilities for x-ray and biopsy to poor patients, taking care of patients refused by hospitals, sending them again to hospitals after they register some improvement, and then documenting their progress.

Through the School of Tropical Medicine, we could successfully contact the Dean of Calcutta University, Faculty of Medicine and famous pathologist, Dr. Subir Dutta who initially used to behave very rudely with me. One day while I was leaving, he threw an indulging smile and said: ***Come and meet me tomorrow. I like to talk to you on what you are trying to do.*** The very next day I went to him. He said: ***You are doing nothing wrong. I am already and shall always be with you. Once I was engaged in a work with a very famous person but he was very emotional and soft-hearted person and I had to pay very high price for his being so. What has impressed me about you is that you are not at all an emotional fool and rather quite tough at heart, though apparently it seems that you are a very soft person.*** He then asked me: ***Have you heard about the Science Congress to be held in Rajasthan this year. Do you know who the Chairman of the Medical Session is?*** I replied by saying no. Another doctor, seated in front of Dr. Subir Dutta, asked me: ***Have you ever heard of the Science Congress?*** I said: ***Yes, I have heard about it but I am not aware of the details.*** They explained to me about the Science Congress. Prof. Dutta had got a proposal to present a paper at the Rajasthan Session of Science Congress. He forwarded this message to me. Prof. Dutta informed me: ***Documenting of a scientific paper in the Science Congress session is a challenging work. This year no paper on cancer is getting presented. If your paper gets selected, then you would be representing Bengal. Again only MDs are allowed to present papers. Since you are not an MD it would be very tough on your part to convince the medical faculty there to publish your work. If you succeed, all your hard work would be paid for. Somewhere I believe, you will be able to achieve this as you have come a long way in this short span of time. You will always find me supporting you from behind. I know this might seem impossible to you now, however, if you can manage to prepare the paper under the guidance of the School of Tropical Medicine and present it, then it will turn out to be a remarkable achievement.*** I thought, I did not possess the minimum qualification needed to have an entry there. I was told that someone would be there that very year, who had once been very close to me and loved me a lot. I was curious to know who he was. Prof. Dutta said: ***Not today. I shall tell you about him tomorrow. Are you interested to know about him just now?*** I said: ***No, since you assured me of disclosing it tomorrow.***

The next day he told me about the presence of Dr. A.K. Hati in the Directorate of Science and Technology as a member of the Program Advisory Committee, with whom I had worked in the School of Tropical Medicine. I was very happy to hear about him. Prof. Dutta asked me first to have a talk with Dr. Hati, and thereafter he himself would talk to him. He further assured that being the Dean of Medical Faculty, he would not allow any genuine original work on Cancer to get erased off. He advised me to work on my paper, as he would be with me. He asked: ***Have you prepared any scientific paper ever before?*** I said that I had

worked with Dr. Manju Ray in the Indian Association for the Cultivation of Science, Kolkata for two years, but within a short span of time, it was really difficult for me to shape out the Abstract, Summary, Materials, Method, Discussion and Conclusion part of a research-paper. Dr. Subir Datta asked me to come every evening at 6 PM. He said: ***Papers have to be written in proper format, because without scientific papers it is difficult to continue with such innovative work.*** Dr. Hiranmay Mukherjee and Dr. Partha Banerjee were two other doctors with whom I had worked in the School of Tropical Medicine. Both of them along with Prof. Subir Dutta, Prof. Amiyo Kumar Hati and Dr. Pranab Bhattacharya rendered unstinted help and cooperation to me. Dr. Bhattacharya was very close to Prof. Subir Dutta. In due course, we showed our paper to Dr. Hati and he approved the work, but he also said that slides would be needed for the presentation. I did not know how to make slides, and I had to find out a way for this. Bhanubabu's shop near Park Circus area, Kolkata, was famous in 90s for preparing slides. He asked me about the doctors who had cooperated with me, and when I named them, he showed profound respect towards Prof. Hati and Prof. Subir Dutta. Ultimately the paper was presented in Indian Science Congress of 1994 titled 'The Treatment of Cancer – A step forward'. The then Secretary of Directorate of Science and Technology, Prof. Dilip Basu, extended all the help in this regard. Each and every member of the New Resource played a significant role in making my humble dream to see the light of the day come true. All arrangements for my journey, food, and lodging in Rajasthan were organized by Sadhubaba who lived at 'Kharagpur', West Bengal, at that time. I am extremely thankful to him.

Previously it had been purely a social work for us, but of late it turned into a scientific research and work for a positive cure. Gradually we felt the need for replacement of the non-technical people of New Resource, by those who were attached to Oncology Forum. There was a crying need for Oncologists and Onco-Surgeons. There we began to search for Oncologists from different corners to form the forum. Right at that time we got to know about Prof. Subir Ganguly and Prof. Prabir Sur through one Prof. Parthajit Banerjee and another, Shri Sourav Chakraborty, and also Dr. Anup Majumdar. Our aim was not fulfilled despite our cordial relations with Prof. Prabir Sur. Prior to this, I had come in close contact of two Cancer specialists like Dr. R.N. Brahmachari and Dr. Chandra (Surgeon, Chittaranjan National Cancer Institute). While working with them I acquired some practical knowledge about Oncology. But still it was not possible for me alone to form an Oncology Forum. Prof. Subir Ganguly, then President of Indian Medical Association, was a reputed personality on Oncology, and gradually a very cordial relation developed between us.

On a few occasions, Oncologists and Onco-surgeons try to show that whatever they think is the best for such patients. Once I came across a well-known Oncologist who used to believe himself as the only chemotherapist in West Bengal, and the rest as radiologists. I met that person at the conference of International Science Congress in Delhi a few years back. He cynically asked me: ***Who are you? Why have you come here with such misleading presentation? Do you know me?*** I replied: ***No, Sir, but I know a few well-known chemotherapists.*** To this he instantly shouted: ***I am the only chemotherapist and the rest are radiotherapists. Who I am you will come to know in the seminar-hall itself.*** One of my friends, Dr. Sanjay Paul, kept on escorting him throughout the seminar. He also tried to influence me but I ignored.

While I was giving my presentation, one of the world-famous oncologists sitting in the judge's row kept quiet for a long time and as I ended, he did not clap at all, and commented that my presentation was of mediocre standard and not up to the mark set for such international seminars. The other judge in the panel said that mine was the most mediocre one and added: ***We had to wait for a long time for this kind of mediocre work! Any such original work can never be up to the mark in the very first attempt.*** To this everybody clapped. As soon as I got down the dias, the same world-famous oncologist hugged me and said: ***I was testing you. I hope, you are not angry with me.*** I could not utter anything but with a deep pain in my heart I simply smiled. Later we became very good friends. Once I asked him: ***Why do you prescribe such expensive chemotherapies to the patients?*** To this he replied: ***I am a doctor, not a social worker. I go by a protocol and do my duty for the well-being of my patients. If the patients think that I am expensive, then they are at liberty to go to some other doctor for their treatment.*** His statements startled me at first but later on I realized that he was not completely wrong. No doubt he is a very good physician (oncologist). He was honestly talking facts.

Around this time, Shri Shankar Sen, then Hon'ble Minister, Prof. Dilip Basu, then secretary of Directorate of Science and Technology, Government of West Bengal, and Prof. Subir Dutta helped me publish another research paper of mine, which was documented by the Calcutta University. This is the paper which showed how a Cancer patient survived under my treatment and care. I have provided the details of that paper in this volume.

Around this time, a paper written by me was presented before the World Assembly on Tobacco Counters Health Forum through this NGO, i.e. New Horizon Centre for Cancer Research and Treatment (New Resource renamed). This paper was later published in Tobacco Counters Health, Vol. 2, in the name of 'Psorinum Makes a Major Breakthrough in the Treatment of Tobacco Related Lung Cancer'. Then on 11th March, 2004, another paper was presented at a conference of 3rd World Assembly on Tobacco Counters Health, and later published in the Tobacco Counters Health, vol. 3, named as 'Non-Conventional Treatment of Tobacco Related Cancer Gradually Gets Right Perspective Through Psorinum Therapy'. One-and-a-half years later a new paper was published in Asia Pacific Journal on Cancer Prevention (Vol.6 No. 2, 2005) on behalf of New Horizon Centre for Cancer Research and Treatment, 381, S. K. Dev Road, Lake Town, Kolkata — 48, West Bengal, India, authored by me along with other doctors under the name of "Attitude of Patients to alternative Medicine for Cancer Treatment". All the papers mentioned above were highly appreciated in various international Science Congress and the treatment of Cancer through this non-conventional method gradually became popular and well accepted, day-by-day earning people's unshaken confidence.

In the 1994 Rajasthan session of the Indian Science Congress it had already been documented that in the case of a stage-IV cancer patient, the disease had completely regressed under my treatment. By the year 1995, the Calcutta University and the West Bengal Directorate of Science and Technology also considered this case to be a success and recognised my efforts. However, the Indian Scientific Committee took a little more time to acknowledge the same. Generally, Scientific Committee utilizes the scientist for the greater good and I was not an

exception. Most of the scientific-committees always fear that its valuable research work might get stolen. They try to protect it by various legal means. On the other hand, I wanted someone to rob me of all my research works. But unfortunately nothing such has happened yet.

At a later stage the IMA (Indian Medical Association, Kolkata) also recognized our efforts. Gradually as we moved forward we started receiving the heat of strong resistance from the Oncology Forum who refused to give any recognition to our work. But I knew that we need their approval so that we could continue with our work. During that time Prof. Partha Banerjee, Dr. Hiranmay Mukherjee and Mr. Saurav Chakraborty brought me in touch with Prof. Subir Ganguly and Prof. Prabir Sur. Prof. Subir Ganguly had for a long time been trying to establish a Cancer NGO. As I was having a first-hand experience, I extended my helping hand and in our house at 381, S. K. Deb Road, another NGO was founded with the name, “ONCOLINK”, and in no time all my works got somehow linked to this organisation. This gave us a whole new platform and under the banner of “ONCOLINK” I got published a few more papers.

Stumbling With Some Stalwarts In The Journey

MAJOR GENERAL DR. AVINASH K. VARMA:

Today when we look back, we see that many influential, intellectual and philanthropic people whom I had come across in my journey and who had guided me at every bent or turn of the road are no longer amongst us. I would like to express my heartfelt gratitude to Late Major General A. K. Varma who was a world famous Cancer physician, scientist and former surgeon to the President of India. He always used to tell me: ***You are bearing a huge responsibility on your shoulders and you have to work very hard to lift the flag in the world’s mega forum. Since the time you started your war against the enemy of health and humanity of the entire society, I have come to know about you from several sources. I have analysed your efforts and come to the conclusion that you are like a soldier. A soldier always moves forward carrying the National Flag, with so many life-threatening risks at every step while carrying the national flag. You do maintain the same attitude and you will see no barrier can ever stop you.***

Once Dr. A. K. Varma became very surprised at and expressed his irritation towards me 16 years ago from now. Around 2003 he told me: ***I have documented all your research and studies and created a scientific platform for you. Why did you not utilize it? Prof. Ganguly, then Director of ICMR, and Dr. Kishor Choudhury, an important office-holder of Scientific Committee, tried to persuade you to take up an important project. But you did not respond to that offer. A number of institutions you had worked with wanted you to head several ventures, but you escaped those as well. I need an explanation: why? You come to various seminars, make sure your work is well published, present the paper somehow and then quietly leave.***

To all his questions I replied back by saying: ***I need to move at a very fast pace. If I take up a project, then I have to work under someone’s guidance. The rules would have been set by somebody else. It is very important for the other person to understand me, my passion***

and my vision.

At that time there were a very few people who understood me and the complex subject I was dealing with. There were so many things involved in my work and it was very important to carve out a unique path that would help to resolve the entire issue by bringing the total problem to a single point solution and then to attack it. Working on a different project could have tied me by many constraints and restraints, and could have well disrupted my speed.

DR. STHABIR DASGUPTA:

I had a very cordial relation with Dr. Sthabir Dasgupta. Though he was not an MD in Radiotherapy, however, later on he turned out to be one of the most renowned oncologists of our time. His contribution in the field of Cancer is exceptional and at the same time inspiring to many. I did not know him from the beginning. In my early life when I was searching for my aim in life, I came across one of his articles in a weekly newspaper and that gave a whole new direction to my life and my approach towards Cancer as a social issue got completely changed.

DR. SAROJ GUPTA:

I am talking here about the time prior to the resistance I faced from the Oncology Forum during early 1990s. At that time the famous oncologists who were engaged in the field of cancer treatment were Prof. Prabir Sur, Prof. Subir Ganguly, Prof. Anup Majumdar, Prof. R. N. Brahmachari, Prof. Amiyo Majumdar (surgeon) and Dr. Saroj Gupta. Here I would like to mention more about Dr. Saroj Gupta, the then Director of Thakurpukur Cancer Hospital, and about how I could come very close to him. There was a time in my career when I was treating a few of the cancer patients who had been turned down by Dr. Saroj Gupta. But soon I faced a very tough situation while treating a lady belonging to an influential family of Kolkata. She was 35 year old suffering from ovarian cancer and ascitis, and was also having some breathing problems. For past several years I had been continuously working on lung cancer and also achieved some success in controlling its effusion. All the patients that I had been treating at that time were all critically ill as well as poor, but showed marked improvement in their condition due to my therapy. Since I had effectively treated the patients suffering from stomach and liver cancer, lung effusion and ascitis, I thought I would be able to handle this particular case as well. But my perception went wrong. Unfortunately, the 35-year-old patient's condition started deteriorating after just one month of treatment. The patient was also receiving treatment from Dr. Saroj Gupta at the same time. One day the patient's family asked me to visit their residence and I met Dr. Gupta face-to-face there itself. I realised that my presence offended him, and he took it as a contempt. He hardly thought well of me and considered me having a marked political background. He informed me that he had decided to administer chemotherapy on that patient followed by a surgery. He questioned me how could I risk the patient's life. One of the patient's family members was a lawyer who threatened to take legal action against me. Later he asked the patient's family whether I had charged any money for the treatment or not. They informed him that I did not ask for even the conveyance cost from them. Dr. Gupta said he already knew a bit about me but also believed that I had marked political connections and the people I used to meet were not a good company to have.

About three months later, Dr. Gupta invited me to his clinic at Dharamtala Street and warned me to stop at whatever I was then upto. Dr. Gupta was a very headstrong man and was against any kind of political influence. He told me that the 35 year old patient had already undergone three chemotherapy sessions and soon the surgery would be performed on her. He also said, he knew that I did not charge any money for treating patients. But Dr. Gupta did not know about other two of his patients who he had refused to attend further, namely, Smt. Binapani Sarkar whose condition improved a lot under my treatment, and another lung cancer patient. Both the patients under my treatment were showing slow but steady recovery. However, suddenly the lung-cancer patient expired, although Smt. Binapani Sarkar's condition showed much improvement. After six months I went to Thakurpukur Cancer Hospital to meet Dr. Saroj Gupta, but he refused to meet me, and asked me not to pay any visit to him in future also. After three days I wrote a letter to him stating that I wanted to discuss the case of Smt. Binapani Sarkar with him and sent him the letter through his P. A.. This time Dr. Gupta somehow agreed to meet me and when met, asked me in a very disgusted manner what I wanted to say. I tried to remind him about this patient whom he had refused to treat. Dr. Saroj Gupta did not recollect about the patient, Smt. Binapani Sarkar, but after some time nodded his head and acknowledged the patient suffering from liver cancer. I told him that the patient was under my treatment and was improving steadily. I also asked him to go through the diagnostic test reports of the patient to identify whether she had cancer anymore or not. Dr. Gupta went through all the documents and remained silent. I told him that I had done everything fairly as far as possible in my capacity to treat her. I used to spend days at her residence in Belghoria and had put my heart and soul together in the case. I also confessed that it had been a mistake treating the ovarian cancer patient without having any experience in treating those kind of patients. Dr. Gupta smiled and told me that I should not blame myself or regret. He then asked me about the correct procedure of treatment for cancer. I replied, 'Chemotherapy', 'Radiotherapy' and 'Surgery'. He then said: ***To treat cancer one should have a fair knowledge regarding all three of them. If you have some knowledge regarding chemotherapy, you would realise that it is very effective in the treatment of ovarian cancer, though it does not provide impressive results for liver, gall-bladder, lung cancer and ascitis. He advised me to keep this thing in mind and also to be careful in future while treating these kinds of patients.*** He shook hands with me and said that he had misunderstood my intentions, and assured me of all help whenever needed by me. I returned back home with a huge peace of mind.

Smt. Binapani Sarkar's case is documented in the paper titled 'Non-conventional Treatment of Tobacco Related Cancer Gradually Gets Right Perspective through Psorinum Therapy' which I have provided at the beginning of the book.

As history repeats itself, after so many years a new cancer case refreshed my memories of Dr. Saroj Gupta. I would like to talk here about a 69 year-old patient, Mr. Sujit Kumar Pal. Mr. Paul was admitted to a local nursing home complaining severe abdominal pain and vomiting. The sonography report indicated gall bladder mass, hepatic infiltration and spread in the liver. He was referred to the Thakurpukur Cancer Hospital. The patient was admitted there under Dr. Saroj Gupta's son, Dr. Arnab Gupta. Dr. Gupta is now the Consultant Surgical

Oncologist and Director of Saroj Gupta Cancer Centre and Research Institute. Mr. Paul was also suffering from gastric obstruction because of which he was not able to eat anything. Mr. Paul found out to have a gall bladder mass on investigation, and FNAC not being suggestive, was decided to post for exploratory Laparotomy and proceed. He underwent surgery on 4/9/2013. The findings showed the mass to involve porta with distal Stomach and GB. Biopsy was taken from liver modular adjacent to GB. Also Gastrojejunostomy and Jejunostomy were done. The findings diagnosed GB mass under investigation (frozen section and imprint cytology positive for malignancy). Malignancy was also found in Tissue biopsy report. The surgery further indicated the GB mass infiltrating liver and duodenum. The patient was suggested to undergo Chemotherapy. But due to the patient's poor health at that point of time, Chemotherapy could not be continued.

The patient visited our facility on 15/09/2013 for the first time but refused to get admitted at that time. May be, he had started losing faith in the available ongoing cancer treatment methodology and before getting admitted wanted to spend some time with his family back at home since he would not be there for a long time with them. He promised me to be back within a few days. He took our drug back home and was taking it regularly. Within a couple of days he started feeling a bit better. He got admitted in our facility-centre on 21/10/2013 under my supervision and also under the constant guidance of Associate Prof. Dr. Amitabha Chakraborty, Dr. Hiranmoy Mukherjee, Dr. Aradeep Chatterjee and Dr. S. Ghosh. On 22/10/2013 a CT Scan of whole abdomen was conducted from Eco- Medical College. The CT features were suggestive of: a) Hepatomegaly with decreased attenuating lesion in right lobe; b) Irregular thick-walled GB with soft tissue mass; c) Nodular lesion in epigastric region. The review study showed partial regression of gall bladder mass since last study of 16.8.2013. Two units of blood transfusion and two units of human albumen were also administered. Gradually the patient's condition improved. After a few days the patient was discharged. We observed significant recovery in the patient's health from the middle of 2015. The patient was again admitted at this facility on 21.2.2016 to undergo a detailed full time observation. Dr. Aradeep Chatterjee, Prof. Anup Majumdar, Dr. Hiranmoy Mukherjee and Dr. A. Goswami decided to conduct another CT Scan of the whole abdomen from Eco-Medical College under Dr. Anup Sadhu, a renowned radiologist and social worker. The CT Scan report showed no hepatic lesion. GB (gall bladder), as visualized, was normal, and suggested clinical correlation and further investigation if clinically indicated. PET Scan was also performed which showed no definite evidence of any recurrent malignant disease in the gall bladder and adjacent liver parenchyma. Mild FDG (F-18 fluorodeoxyglucose) uptake was noted in the pylorus of stomach with no obvious anatomical abnormality – likely physiological. There was no evidence of any locoregional lymph nodal and distant metastasis noted.

Critical Cancer Management Research Centre & Clinic
(Run by Dr.Ashim Chatterjee & Other Renowned Oncologist & Onco Surgeon)

3381, S.K.DEB ROAD WEST BENGAL (INDIA) KOLKATA-700 048



REGISTRATION SHEET

Regd, No..... 876/13-14
Date..... 21-10-13
Patient I.D.No.....

Name of Patient Mr./Ms. Singh, Mrs. K.D.
Age 69 Sex Male Religion Hindun
Name of Father/Husband Late B.B. Dab
Address Abundant Boudgopora ward no. 2, New - New Amarsar Road
Amargarh
P.S. Amargarh Dist. Medinipur PO. Amargarh
Tele No 0382-225586 Mobile No. 97941888
Admitted by Dr. Suman Ghosh
Address D/o Tele No. _____ Mobile No. _____

Name of Doctor..... Tel No.....
Case.....
Admitted on. 8/10/46..... at 12.55.....
Discharge on. 05/3/20/46..... at 7.30 AM.....

.....
Signature of Guardian



SARC, GUPTA CANCER CENTRE & RESEARCH INSTITUTE

formerly known as
CANCER CENTRE WELFARE HOME AND RESEARCH INSTITUTE
Mahatma Gandhi Road, Thakurpukur, Kolkata - 700 063, India

Ph: 91-33-2453-2751 / 82 / 83, 91-33-2467-8001/8003, Fax: 91-33-2467-8002, 91-33-2467-8003
E-mail: cancercarewelfare@gmail.com / cancerwelfare@yahoo.co.in, Website: www.cancercentreindia.org

Patient's Name: SUJIT KUMAR PAL

Regn. No. 201305517G

Date of 1st Attendance: 23/08/13

Age: 66 YRS.

Sex: MALE

Name of the father/spouse:

Visiting Doctor: DR. A. GUPTA

Dept. SURGICAL ONCOLOGY

Ward: DR.

Cabin/Cubicle/ITU/General Bed No: 30

Date of Admission: 31/08/13

Date of Discharge: 14/09/13

Summary :

Mr. Pal found out to have a Gall Bladder mass on investigations, and FNAC not being suggestive, was decided to post for exploratory laparotomy & proceed. He underwent surgery on 04/09/13. Post operative recovery is good & he is being discharged in a stable condition.

Final Diagnosis :

GB Mass under investigation (frozensession + Imprint cytology positive for malignancy).

Treatment Summary :

Surgery :-

Name of Surgery: Open Biopsy from liver +

Date: 04/09/13

Gastrojejunostomy +

Jejunoejunostomy done on

Diagnostic laparoscopic done

-No liver nodule or peritoneal

deposit found

Abdomen opened with subcostal &

Type of Anaesthesia: Under GA.

Findings: Mass involve porta with distal stomach & GB.
Biopsy taken from liver nodular by adjacent to GB.
Gastrojejunostomy + Jejunoejunostomy done.

Closure: Wound closed with 1-0 PDS + skin stapler.

Page 1 of 3

Donations to our hospital are exempted under Section 80G, and for our Research Activities under Sec. 35(1) of Income Tax Act.



SANDIP GUPTA CANCER CENTRE & RESEARCH INSTITUTE

FORMERLY KNOWN AS

CANCER CENTRE WELFARE HOME AND RESEARCH INSTITUTE

Manasmita Gandhi Road, Thakurbukur, Kolkata - 700 063, India

Ph : 91-33-2453-2761 / 82 / 83, 91-33-2467-5001/8003, Fax : 91-33-2467-5002, 91-33-2453-47

E-mail : cancercentre8@gmail.com / cancerwelfare@yahoo.co.in, Website : www.cancercentrecalcutta.org

Signature of Doctor: _____

Name (Block Letters): DR. N. SAHA

Designation: R.S

Department: SURGICAL ONCOLOGY

Date: 14/09/13.

1. I have received all the belongings of my patient together with the reports of investigation, Radiological plates and all papers brought by us to my entire satisfaction.
2. We have been fully explained the condition and prognosis of the patient and taking the discharge accordingly.

Signature / LTI of Patient : _____

Name of patient : _____

Signature of Relative : _____

Name of Relative: _____

Relation with patient: _____

Date : 14/9/13

Time : 12:30

Address : _____

Dr. - Pallab Kumar Mondal
Fig-721801.

SUJIT KUMAR PAL

66 YRS

22.10.2013

DR. OF MCH

16810/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient of Ca. gall bladder with hepatic involvement – last CT 16.08.13.

TECHNIQUE

Plain, oral and I.V. (non –ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no detectable abnormality.

Liver is enlarged with decreased attenuating lesion in right lobe of liver.

Gall bladder is irregular thickwalled with soft tissue mass inside.

Common bile duct is not dilated.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

SUJIT KUMAR PAL

66 YRS

22.10.2013

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Prostate appears normal in shape, size and contour. There is no focal lesion in it. No evidence of prostatic calculi. Periprostatic fat planes are normal.

Nodular opacities seen in epigastric region anterior to stomach.

Lung bases are clear.

IMPRESSION

CT features are suggestive of :

- 1) Hepatomegaly with decreased attenuating lesion in right lobe.
- 2) Irregular thickwalled gall bladder with soft tissue mass.
- 3) Nodular lesion in epigastric region.

Review study shows partial regression of gall bladder mass since last study of 16.08.13.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD

DR.ANUP SADHU
DMRD, MD.

7/19/2016

img098.jpg



CANCER CENTRE WELFARE HOME AND RESEARCH IN

Mahatma Gandhi Road, Thakurpukur, Kolkata 700 063

Phone : 2453-2781/2782/2783, 2467-8001/03, Fax : 2453-6711, 2467-800.

E-mail : ccwhri@cal2.vsnl.net.in / cancerwelfare@yahoo.co.in Website : www.cancercen

REF. NO. : I-58

DATE OF RECEIPT : 04-09-

PATIENT : Mr. Sujit Kr Pal (56 YRS.)

DATE OF REPORT : 09-11-

13/5517
CSB-UP-28.

Referred By : Dr. A Gupta

HISTOPATHOLOGY REPORT

Specimen : Wedge biopsy from liver.

Gross : All tissue embedded.

Micro : Sections studied show features of metastatic adenocarcinoma.

Slide No. : H-7215/13.

PROF. SUBRATA CHAKRABORTY,
MD (PATH).
CONSULTANT PATHOLOGIST.

*Case seen in
consultation with*
DR. ANURADHA DE
MD (PATH), AIIMS
PATHOLOGIST

Dr
DR. SUPTI MUKHOPADH
MD (Path)
CONSULTANT
PATHOLOGIST

Super



SUJIT KUMAR PAL

66 YRS

23.02.2016

DR. OF MCH

22467/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient Ca. GB with hepatic infiltration - operated on 04.09.13. - had (GJ) jejunostomy.

TECHNIQUE

Plain, oral and I.V. (non -ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no detectable abnormality.

Liver is normal in size & density. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Porta hepatis appears normal.

Gall bladder as visualized shows clear lumen.

Common bile duct is not dilated.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd



SUJIT KUMAR PAL

66 YRS

23.02.2016

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Prostate appears normal in shape, size and contour. There is no focal lesion in it. No evidence of prostatic calculi. Periprostatic fat planes are normal.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

GB is adhered to distended stomach.

IMPRESSION

Present CT scan of abdomen shows no hepatic lesion.

GB as visualized is normal.

Suggested clinical correlation and further investigations if clinically indicated.

DR.D.SHARMA
MD

DR T.K.DHAR
MD (Radiodiagnosis)

DR.ANUP SADHU
DMRD, MD.

AT Medical College & Hospital Campus

88, College Street, Kolkata - 700 073, Phone : 2212-3778/3779

Identity of the patient not verified, if there is any lack of correlation between the result and clinical condition, please refer the patient to the respective department.

SALIL GUPTA CANCER CENTRE & RESEARCH INSTITUTE

CANCER CENTRE WELFARE HOME & RESEARCH INSTITUTE

Maratha Gandhi Road, Thakurpukur, Kolkata - 700 063

Phone: 2467801/82/83, 24678001/03 Fax: 24678002 / 24536710 / 6711

HISTOPATHOLOGY REQUISITION FORM

Date of Receipt.....

Slide No.

Ref: Mr./Mrs./Master: Sig: / - 557 - Pad

Age: 56 Regn. No. 13/5517- Date of Admission.....

Ref: Dr. Gupta Date of Operation..... Bed No. 55017

Signature.....

Nature of Specimen:

Clinical Details	Previous FNAC/HP	Diagram
G.A. mass infiltrating Liver & Duodenum (FNAC non diagnostic)	→ wedge biopsy from liver → frozen section	

GROSS: F-10 / 13

- A) General Description: Sections reveal fibrocollagenous tissue fragments focally infiltrated.
- B) Tumour - Size: by neoplastic cells with glands lined by atypical cells
- Cut Surface: by atypical cells
- Others: Imp: - Positive for malignancy

C) Margins:

D) LYMPH NODES:

E) OTHERS:

Grossed by.....

Date.....



EKO PET - CT & DIAGNOSTIC INSTITUTE
(A UNIT OF EKO DIAGNOSTIC PVT. LTD.)

DEPARTMENT OF PET CT

Patient Name : Mr. SUJIT KUMAR PAL
Age / Sex : 69 Yr(s) / Male
Ref. Doctor : Dr. ASHIM CHATTERJEE
Lab No :

Reg No / Visit No : 03-15-001415/1
Requisition No : 03-RQ-PE-15-000621
Requisition Date : 29-Feb-2016
Report Date : 29-Feb-2016

CLINICAL HISTORY:-

Follow up case of carcinoma of gall bladder with hepatic infiltration; post resection and gastrojejunostomy and jejunoejunostomy done on 4.9.2013.

PROTOCOL:-

Whole body PET-CT scan was done after I.V. injection of 10.9 mCi of 18F-FDG, using a whole body full-ring dedicated Biograph mCT 128 slice PET-CT scanner. CT images were obtained using Care Dose 4D and Care kV. CT based attenuation correction was done. Images were reconstructed using True X (HD) and reformatted into Transaxial, Coronal and Sagittal views. A 3D image and fusion images of PET & CT were obtained. Oral and intravenous contrast enhanced CT scan of brain, neck, chest and whole abdomen was also performed.

A semiquantitative analysis of FDG uptake was performed by calculating SUV values corrected for dose administered and patient body weight. Fasting blood glucose-113 mg/dl, Serum Creatinine-1.70 mg/dl and FDG uptake time-79 mins.

FINDINGS:-

Physiological distribution of metabolism noted in the bilateral hemispheres of cerebrum and cerebellum.

Nasopharynx, Oropharynx, hypopharynx, parapharyngeal region and larynx appear normal with no abnormal FDG uptake seen in relation to them.

No enlarged cervical lymph node with increased FDG uptake is seen.

Thyroid gland appears normal with no abnormal FDG uptake.

The lung parenchyma shows normal bronchovascular pattern. No pulmonary parenchymal lesion with increased FDG uptake is seen. There is no evidence of pleural effusion.

No enlarged mediastinal lymph node with increased FDG uptake is seen.

No abnormal FDG uptake/mass lesion noted in the gall bladder. Gall bladder is in close relation to the distal stomach likely due to adhesions.

Liver, gall bladder, spleen, pancreas and adrenals appear normal with no abnormal FDG uptake seen in relation to them.

There is no ascites.



EKO PET - CT & DIAGNOSTIC INSTITUTE
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DEPARTMENT OF PET CT

Patient Name : Mr. SUJIT KUMAR PAL
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Mild FDG uptake noted in the pylorus of stomach with no obvious anatomical abnormality (SUVmax-5.26).

Evidence of gastrojejunostomy and jejunostomy noted.

Physiological bio distribution of tracer noted in the bowel loops.

No enlarged abdominal lymph node with increased FDG uptake is seen.

Physiological bio-distribution of FDG noted in kidneys and urinary bladder.

Non FDG avid cystic lesions noted in bilateral kidney (largest- 2.75x1.75 cm) – simple cysts.

Prostate appears normal with no abnormal FDG uptake.

No focal lesion/abnormal FDG uptake is seen in skeleton.


IMPRESSION:

PET-CT study shows no definitive evidence of any recurrent malignant disease in the gall bladder and adjacent liver parenchyma.

Mild FDG uptake noted in the pylorus of stomach with no obvious anatomical abnormality – likely physiological.

No definitive evidence of any locoregional lymph nodal and distant metastasis noted.

Dr S K SHARMA
MD
DIRECTOR


Dr G RAMA MOHAN REDDY
MD, Nuclear Medicine (AIIMS)
CONSULTANT & HEAD
PET-CT & NUCLEAR MEDICINE



SAROJ GUPTA CANCER CENTRE & RESEARCH INSTITUTE

formerly known as

CANCER CENTRE WELFARE HOME AND RESEARCH INSTITUTE

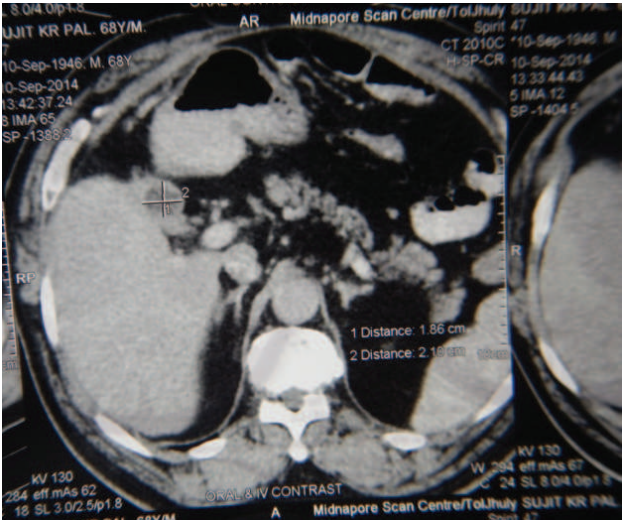
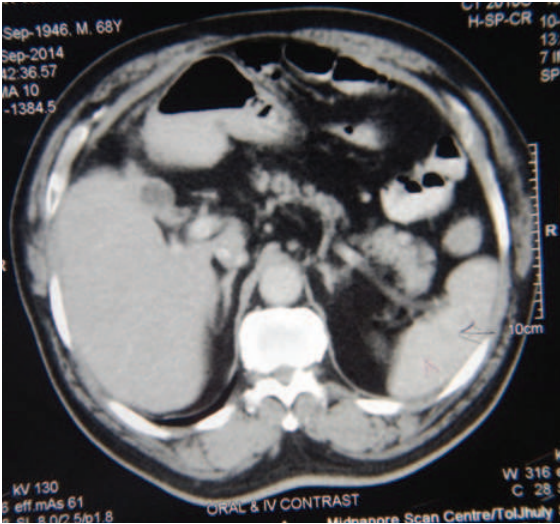
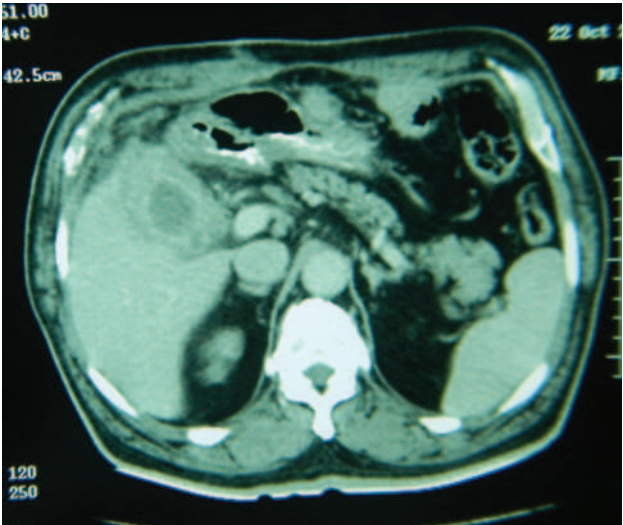
Mahatma Gandhi Road, Thakurpukur, Kolkata - 700 063, India

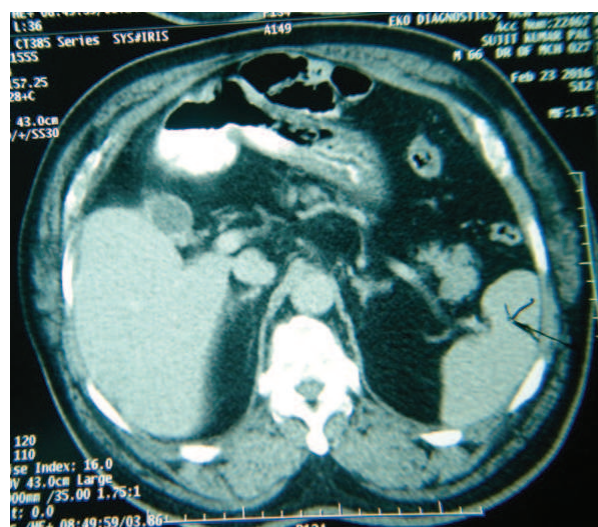
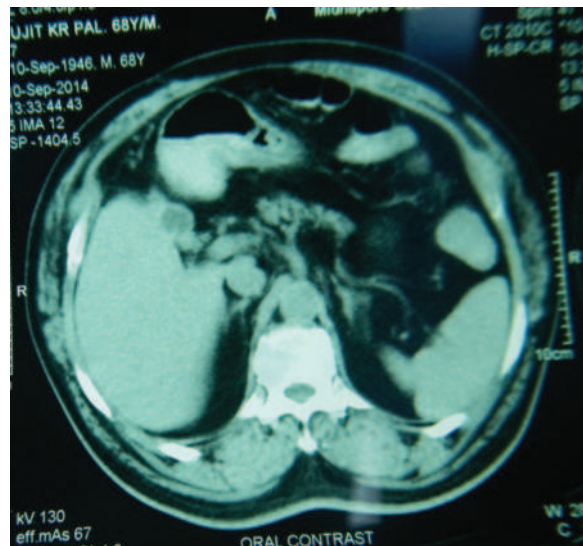
Ph : 91-33-2453-2781 / 82 / 83, 91-33-2467-8001/8003, Fax : 91-33-2467-8002, 91-33-2453-6719

E-mail : cancercentre@gmail.com / cancerwelfare@yahoo.co.in, Website : www.cancercentre.org.in

Advice on Discharge :

- * Nutritious diet
- * Cap. Propan DSR - 1 cap OD before breakfast x cont
- * Syr. Cremaffin - 4 tsf after dinner x cont
- * Protal - D - 4 tsf BD x cont
- * Tab. Aminos - 2 tabs TDS x 10 days
- * Syr. Dexorange - 2 tsf BDPC x cont
- * Tab. Urimax 0.4 mg OD x cont
- * Tab. Telvas 50 mg OD x cont
- * Tab. Daxid 50 mg OD x cont
- * To attend SOPD on next Friday 20/09/13 with Biopsy report for follow up & further management.





Slides of Sujit Kumar Paul



Sujit Paul



Sujit Kumar Paul with Prof. Jaydeep Biswas and Dr. Asim Chatterjee



Sujit Kumar Paul with Prof. Jaydeep Biswas

Dr. Saroj Gupta was a great man. I call him great because he had guided me during a very crucial phase of my life. He advised me to learn mainly Chemotherapy and Radiotherapy in detail. He told me that I would have to know things better than anybody else because at that point of time doctors were following the conventional treatment methods and in order to excel over them I would have to prove the effectiveness of my drug and treatment methodology. When Dr. Arnab Gupta's patient came up to me, I thought if, by any means, the patient could be saved just like Smt. Binapani Sarkar's case, it would be a tribute from my side to Dr. Gupta who was unfortunately no longer with us. A very important role was played by my son, Dr. Aradeep Chatterjee, in the critical treatment and recovery of the patient, Mr. Sujit Kumar Paul.

PROF. GAURIPADA DUTTA:

There was a time when people who were actively taking care of the positive treatment of cancer and were finding out new ways to tackle the problem, they were the very few eminent personalities who used to be politically motivated as well. I decided to work under the guidance of a few of them. Among them, one was Prof. Gauripada Dutta.

One day I went to visit Prof. Gauripada Dutta accompanied one Bikash Dutta at his residence. His first reaction was: *I know everything about you and your work, both personal and professional. I grant you ten minutes to present your case in front of me. I am granting you this much of time only because you have come here with Bikash. I am surprised at how you have utilized him. He is my relative. The way you have used him, in the same way I have also come to know about your whereabouts from him. Not only me but the entire scientific community is very curious to know what exactly you want and what you are looking for. I have also come to know about you from Dr. Subir Dutta. I do not believe it completely. I am a very busy man. I do not have too much time to waste on you. Ten minutes is what you have. Say whatever you have to say quickly.*

Although Prof. Gauripada Dutta had allotted me ten minutes, the discussion went on for three hours. In these three hours the way he treated me I would like to avoid providing those details here. Then again Prof. Dutta asked me to sit in discussion with him the same night at 9 p.m. and allotted me ten more minutes at his chamber. This session also continued for three long hours. Again he asked me to visit him the very next day at his chamber. At the end of that day's discussion Prof. Dutta said: *I fail to understand what you actually want. From whatever you have said to me, I can figure out that you want to enclose the entire sky in your small bag. Your intentions are not bad. Alright, I assure you that if you ever get involved into any trouble, I will not hesitate to take care of it.*

Research And Treatment: Formation Of New Horizon Centre For Cancer

By the time 'Oncolink' started functioning with its core group of oncologists, I had already taken the initiative to set up a new cancer institute and a research facility. I had decided upon the core governing and executive body of the institute. When I put forward this proposal in front of the Oncolink members and staff they accepted it unanimously. I along

with Prof. Ganguly, Dr. Amit Sarkar and one Manoj babu went to many places in search of land at a proper location for our institute. In the meantime, I received a proposal from a builder. There was a disputed property behind the Ruby General Hospital. He offered us that if we could take care of the dispute and help to legally sell the land, he assured us 5 bighas of land for our cancer institute. We were all very glad. Everything went about as planned. As soon as the land was pulled out of dispute, the entire episode changed dramatically, as they declined to do any favour as promised. We had too many expectations from the project. We felt cheated and disappointed. I thought that my fellow colleagues would be angry over this incident, but nothing such happened. However, my target to build a cancer hospital did not die out. During this time a few difference of opinion came up between me and the members of Oncolink. I also realised that whatever I wanted to do would not be possible during my association with Oncolink and I decided to part ways and communicated my decision to Prof. Ganguly. I told him that I would need to create a new platform which would allow me enough space to do my work freely. Prof. Ganguly asked me if I had thought of something, to which I replied with a yes. Prof. Ganguly said: ***I do not have much objection and have full faith in you. The people you are working with will never support you, and in fact would go against you. They will try to stop you in every way possible. But you have to remain determined and move ahead without paying any importance to them. I don't want you to lose your identity.*** He assured me that he would make the other members of Oncolink also understand. A few months later I formed the NGO, named 'New Horizon Centre for Cancer Research and Treatment', and started working on its development. I had plans to unite both the NGOs. Prof. Ganguly stated: ***We do not always get what we want but I would be able to use both the platforms simultaneously.*** This I was able to achieve and two papers got published under the joint initiative.

While deeply engaged in New Horizon Centre for Cancer Research and Treatment, I got immense support from the then ruling party of West Bengal. They were ready to help me with anything I wanted. At that point of time HIDCO was constructing a township at Rajarhat, Newtown. The chief-engineer of HIDCO at that time was one Mr. Shyamal Bhattacharya whose wife was suffering from advance stomach cancer. They first went to Prof. Ganguly for advice and he referred them to me. Mr. Shyamal Bhattacharya said that his wife was meant everything to him, and their son was at that point of time an engineering student. I started treating the patient and within a few days her condition improved. This is the time I got myself introduced to the then chairman of HIDCO, Mr. Gopal Mukherjee. We knew Minister Gautam Deb for a long time. Surprisingly, two P. A.s' (Prasad Basu and Pranab Dasgupta), one's mother and the other's father were affected with cancer and was under my treatment. Gautam Deb's C. A. Rajiv Biswas's mother-in-law was also suffering from cancer and was getting treated by me as well. All of them stood by us at the time of need. Mr. Gopal Mukherjee committed to us for allotting 1 bigha of land for our facility. He instructed Mr. Shyamal Bhattacharya to stand by my side and to look into everything. During this time a renowned engineer, Mr. Bhanja Choudhury also came to my contact, as I was treating his son who had cancer. He also got involved in the project and planned a 25-bedded hospital and research centre with garden and greenery all around.

My Association With Subodh Mitra Cancer Hospital, Salt Lake, Kolkata

At this point of time when I was seriously engaged in setting up a Cancer Research Centre of our own, I received a request from the President of Subodh Mitra Cancer Hospital and from former minister, Shri Subhash Chakraborty to join the hospital as the Chief Coordinator. I felt, if I accept this offer, bulk of my time would be wasted, while at the same time, the progress of my research would speed up too. Earlier Dr. Ashish Mukherjee and Dr. Advani from Tata Memorial Hospital (Bombay) used to visit the Subodh Mitra Cancer Hospital regularly. But as soon as Dr. Ashis Mukherjee left Subodh Mitra Cancer Hospital, Dr. Advani started making less number of visits. Even then performance of the hospital kept on failing. I thought, something should be done to revive the hospital. A lady doctorate and a well-wisher of mine then advised me to join Subodh Mitra Cancer Hospital. She said that construction of the proposed hospital would take a long time and according to her Subodh Mitra Cancer Hospital would bestow upon all the opportunities I had been searching for so long. According to her, it would make me gain administrative experience also required for running a hospital.

During the period 2002-2003 I got attached to Subodh Mitra Cancer Hospital in Salt Lake and was involved in both clinical and administrative works. The then existing area of land belonging to Subodh Mitra Cancer Hospital were taken on a lease from the West Bengal government on 19/02/1998. The Board of Trustees consisted of nine prominent personalities including Justice (Retd.) Chittatosh Mukherjee, Dr. Jayshree Roy Chowdhury and others.

Just after one year, from 15th April, 1999, the hospital started to move on its path of rendering service to the people. Initially treatment used to be provided only at the external front but within a short span of time all other arrangements were made. In 2000 the Thalasemia Foundation kept some of their instruments in this institution. The hospital authority gave them the permission for both donation and collection of blood for the patients. But since 2001, Thalasemia Foundation got shifted itself elsewhere and as such Subodh Mitra Cancer Hospital began to work in full space.

The Hospital had been under a Trust Body, with particular objectives of carrying out research works inter alia, although till date no life saving-drug for cancer has been visualized. This Hospital, however, always tried its best to provide patients with the minimum treatment required in the last days of their lives. In course of time, it seemed to have deviated from its avowed objectives, and the service remarkably deteriorated because of lack of coordination among the doctors and the administrators.



Subodh Mitra Cancer Hospital, Salt Lake, Kolkata

On 7th November, 2002, many old trustees were replaced by new enthusiastic persons. It was alleged that the previous management could not properly run the hospital on account of their laxity in initiative. After a few days the Board of Trustees appointed a new Medical Director of the Hospital. Gradually number of patients increased and the hospital building was transformed into a five-storied one. The staff members were devoted to the service towards the patients and worked hard. But unfortunately after some time misunderstanding amongst the doctors crept in and their inner conflict came out on the surface. As a result the administrative authority of the hospital was having great difficulty and confusion in running the hospital in the desired manner. Unhealthy ego-clashes and political grouping of the doctors vitiated the hospital-environment. In this chaotic situation I however maintained absolute calm and neutrality and tried to be wholly engaged in my job-schedule only. Everybody was aspirant to dominate the hospital administration and unprincipled groupism was found rampant. On account of my impartial and non-interfering attitude the hospital authority, finding no other alternative, requested me to co-ordinate all the functions of the hospital in a healthy way.



The Subodh Mitra Cancer Hospital

This offer of the position as chief coordinator gave me a unique opportunity to run a cancer hospital of such a great magnitude independently. My dream came true and I accepted

the offer gracefully.

I sat in a discussion with the hospital administration and suggested for immediate renovation of the hospital building. The Board of Directors decided to modernize the hospital and renovate the building. As I was quite eager to work in the terminal centre, so I proposed to the authority to set up the same. In the Board meetings I had to face strong protests and refusals since, at that time i.e. 14-15 years ago, there was no concept of Terminal Centre for Cancer patients in India. It was a foreign concept and they had different sets of protocol. Then Shri Subhash Chakraborty asked me: ***How would you be able to recreate something that is new and foreign to us, and apply such a complex protocol here?*** To this I humbly replied: ***We will not follow the foreign protocol but will develop something of our own. There are no Terminal Centres in India but what we will create will follow the Indian protocol of rationality.*** Shri Subhash Chakraborty became very happy and was found satisfied at my suggestion. This news was published in almost all newspapers. One of the relatives of my patient who had died because of Cancer donated 1 lakh rupees towards the primary expenses of the proposed Terminal Centre. Eventually this Terminal Centre gained popularity. I worked there for long 6 years, and got my experience highly enriched. Eventually the idea of going ahead with New Horizon Centre for Cancer Research and Treatment had to be dismissed as it was created centring me.

With all cooperation from the doctors and staff who acknowledged my efforts, I succeeded in developing a vibrant terminal centre. So far I worked with terminal patients in different institutions in a sporadic manner and not independently. Now my desire was fulfilled to work independently in a terminal cancer centre. It is important to note that the terminal centre of Subodh Mitra Cancer Hospital was the first of its kind in Kolkata and perhaps in India. Gradually the State Health Department agreed to arrange for supply of Morphine to treat cancer patients. To be more specific for me this was the first scope of conducting institutional treatment of cancer by my newly developed therapy, as a result of which I feel no difficulty at present to handle all complex situations in a hospital.

Initially the situation was not so smooth. I was unaware of the thin distinction between institutional directive and treatment procedure. Kind cooperation received from the Secretary of the Hospital and others helped me a lot to overcome the difficulties associated with palliative treatment. Palliative Treatment Ward was built up on 25/12/2004 in the hospital premises with five beds along with modern facilities. A room was provided on the third floor of the hospital which housed idols of deities from every faith in order to cater to the spiritual needs of the patients. This aspect was one of the special characteristics as per international protocol which should be adhered to in case of pursuing palliative treatment in cancer terminal centre. Ample evidences are there how the hospital authority could successfully work in the palliative unit. Till the beginning of 2008 I remained attached to the hospital and treated a large number of terminally ill cancer patients with Psorinum therapy together with palliative treatment. My long-standing dream of administering a centre for terminal patients vis-a-vis running a clinic suitable for their palliative treatment along with a research centre ultimately became a reality. I gathered invaluable experience from working independently as a chief coordinator in arranging seminars in various departments and in performing the job of liaison personnel

of the hospital.

Mr. Subhash Chakraborty bestowed on me a very important position in the Hospital where I was responsible for the total administrative activity of the hospital and was also involved in almost all relevant decision-makings. He is no longer amidst us. However, I will always remember him with profound regards. I used to consider this sudden turn of my fate as an unsolicited blessing, since I lacked in the experience to run a cancer institute independently at that point of time. There remained a huge knowledge and experience gap between my fellow colleagues like Prof. Subir Ganguly, Dr. Gautam Mukhopadhyay (surgeon), Dr. Sharadwat Mukhopadhyay MD, and the like, and me. The only exception was Prof. Rita Chatterjee. I shared a cordial relationship with Prof. Rita Chatterjee and her husband.

Throughout my life I have emphasized on building good relationship with the staff wherever I have worked and in turn they have grown fondness also towards me. In my early life, a few of the oncologists who had guided me were also associated with the Subodh Mitra Cancer Hospital. I have always tried to maintain due respect towards the people who inspired me in the journey to reach my goal. While working at the Subodh Mitra Cancer Hospital, I always tried to let my staff never know what power and responsibilities were in my hands. I shared an amazing relationship with Mr. Subhash Chakraborty. He always used to ask about the progress of my work and also about the hospital. He had huge faith in me and my decisions. I also shared a good rapport with his Secretary and General Manager, Sudeen Bhattacharya. He also used to praise my work a lot and gave appropriate respect to my decisions. I was given special treatment by both of them. I was provided with an exclusive AC chamber, a car for my conveyance etc., but I never misused it. I would never forget the assistance I received from my fellow colleagues. Although I did not become a part of this system in a conventional manner, still I was always open to any suggestion that seemed to have been for the betterment of the hospital. I was at the same time trying to connect Subodh Mitra Cancer Hospital with the whole of South Dum Dum Municipality area for Cancer detection and treatment of the poor at a very nominal expense. Unfortunately, due to some political disputes in that area, this venture could not achieve the desired goal. Not surprisingly, at Subodh Mitra Cancer Hospital a few doctors (non-oncologists) raised their voices demanding that if better results were to be obtained by the use of non-toxic Psorinum therapy without applying chemotherapy, modern management support, anti-biotics, blood transfusion, surgery and the like, then why should chemotherapy be still applied. Except the oncologists, general physicians and our supportive hospital-staff had time and again asked me to combine the Psorinum therapy with the ongoing treatment method which I declined. This dispute gained its ground, but without getting into any kind of confrontation I resigned from the Subodh Mitra Cancer Hospital to avoid any sort of unwanted situation that might affect the Hospital's image. My colleagues who at that time believed that I had been behind the entire controversy, soon realised that I was above board.



Subodh Mitra Cancer Hospital, Dr. Asim Chatterjee's work place as the Convenor

During my tenure at Subodh Mitra Cancer Hospital an interview of mine was published in the Bengali newspaper 'Anandabazar Patrika'. If one goes through it, one will realise what kind of cordial relation and fondness I had grown towards the hospital.

While being at Subodh Mitra Cancer Hospital, Prof. Rita Chatterjee to whom I would always remain obliged, helped a lot me towards my research and study. She too, after resigning from her position, joined the Subodh Mitra Cancer Hospital and for nearly two years supported and advised us on many matters related to the subject. She was basically from the academic background. I had been engaged into many social activities at that time, moving from one village to the other, conducting surveys for Cancer detection, creating Cancer awareness under the banner of 'Roko Cancer' particularly related to breast Cancer in West Bengal. Ms. Rita Chatterjee would always ask me to concentrate on the primary objective than wandering here and there, but soon she too found the situation to be very alarming, and she along with Dr. Banerjee joined us.

In an interview with Dr. Asoke Bose by Piyanka (Piyanka is an M.Sc. in Economics and doing research on backlash of cancer on Indian Society), when asked about his association with Subodh Mitra Cancer Hospital, he replied, "I joined the hospital officially in the year 1999 as a Trustee and as the vice-President. Later on, since 2002 I am holding the position of the Secretary in this institution."



Dr. Ashok Bose

Piyanka then inquired, “In that case you have been a part of Subodh Mitra Cancer Hospital from the very beginning. When Dr. Ashish Mukherjee and Dr. Advani joined the hospital, from that time there was significant development in the hospital. Unfortunately, after they left there was a phase when nothing was going right. Most likely, this was the time when it became very difficult to run the hospital. At this point of time on Mr. Subhash Chakraborty’s insistence Dr. Chatterjee joined the hospital. Soon after that, a few development-oriented decisions were taken and the revival of the hospital could be possible. I have learnt about you from Dr. Chatterjee as well as from Pujan babu, and I wanted to meet you and also to visit this facility as Dr. Chatterjee has time and again stated that this facility and you in particular had contributed significantly in what he has achieved and what he is today. I have also come to know from Pujan babu that Dr. Chatterjee played an untold role in the growth of this hospital and for its popularity among the masses. What do you have to say in this context?” To this, Dr. Bose responded by saying, “Soon after the hospital was set up we started facing administrative as well as financial difficulties while running the set-up. Dr. Jayasree Roy Choudhury wanted to dispose of the facility. At that time we approached Late Subhas Chakraborty. The former Transport Minister financially supported us at that point of time and helped the hospital to regain its ground and also invited Dr. Ashish Mukherjee and Dr. Advani to join the hospital. After they joined we witnessed significant development in the hospital, but it was an unhealthy growth. The hospital used to be very much crowded at that time with too many patients and their family members visiting the facility. We also realised that our objectives were not quite matching. These two doctors were associated with the big multi-speciality hospitals mushrooming in Kolkata at that time. They soon decided to move out of Subodh Mitra Cancer Hospital and thus our association lasted for a short time. This was the time when it became very difficult for us to run the hospital. Also the hospital’s entire responsibility came upon my shoulders and since 2002 I held the position of the Secretary in this institution. Sri Subhash Chakraborty invited Dr. Asim Chatterjee to join the hospital as the Director, but Dr. Chatterjee refused, and accepted the position of the coordinator of the hospital. Although he acted as a coordinator, still his role was not limited to his designation. He was among the major decision-takers in this facility. I also allowed him to do anything he wanted for the benefit of this facility. Dr. Chatterjee always used to look into the constructive development of this facility. In due course of time we realised that he is a skilled administrator and very soon he learnt well the situation here and started looking

for solutions to overcome that scenario and worked towards its stability. He had a far clear perception of which department should continue and which department to discontinue to match the situation. The terminal centre was created on Dr. Chatterjee's suggestion. This centre played a very significant role not only in the revival of the hospital but also benefited many patients and their families, especially those who came from north-east region. Also when there was severe opposition against the setting up of the ICCU and a dialysis unit at this facility and questions arose about its utility, Dr. Chatterjee said that definitely both were required, as these would be the much-needed additional facilities to serve the patients better. The hospital already had a surgical unit. However, Dr. Chatterjee gave a clarification: ***Cancer-affected people always require the ICCU but it is not feasible for all institutes to provide ICCU all the time. To meet extreme emergency, ICCU support is required for critical cancer patients. Besides, some cancer patients require surgical intervention at times, which should be conducted in an ICCU because in most of the cancer patients' cases multiple organs may be found affected. On principle ICCU is required for the cancer patients though the results are not always satisfactory.*** He further added: ***In many cases the excretory system of the patients might get affected like kidney, ureter, bladder etc., and the need for dialysis may arise at times. The results are consistent for temporary period. Until and unless the locus of the disease is removed, results remain highly unpredictable.*** On the other hand, when we asked for his advice to create a Radiation Unit, he simply dissented. He justified his position by stating that the hospital is located in a populous locality. Although he said that he was not an expert on this, still to him this venture was not viable with the few number of beds available at this hospital and with such skeleton staff. Again according to him, installation of a radiation machine would involve many technical difficulties, both civil and mechanical, whenever there were breakdowns.

“Still we went ahead with the idea and a large investment was made. We realised our mistake when necessary permissions to run a radiotherapy unit was denied to us. We had to face severe financial loss as well. The people-oriented work done by Dr. Chatterjee added to the goodwill of the hospital. Dr. Chatterjee also used to decide upon the infrastructure as well as the appointment of staff. He on a couple of occasions arranged finances for the hospital. Again during his tenure he did not even charge a single penny for his services. A person has two lives – his personal life and his work life. Dr. Chatterjee has always maintained a balanced work life throughout. He is such a person that even his enemy would not have any indictment against him. He always has a smile on his face, which is very encouraging. He left the hospital due to a few controversies. This hospital followed the traditional method of treating cancer but Dr. Chatterjee's drug, Psorinum, was also creating its own identity as an alternative drug to treat cancer, which was very effective in the cases involving Glioblastoma multiforme (GBM), lung, liver, pancreas and stomach. Our General Manager, Sudeen Dasgupta's sister was responding very well under Dr. Chatterjee's treatment by Psorinum Therapy. During the entire controversy, the management and a few doctors stood by the side of Dr. Chatterjee. Even then he decided to quit without going into any further controversy. Till date we share a very good relationship and to hear that he has been benefited from this institution i.e. Subodh Mitra Cancer Hospital is a huge honour to us.”

The hospital authority always tried to be compassionate to all the patients and provided them with various kinds of services with least cost. While discharging our duties we have also experienced chaos in the terminal centre due to infectious insects coming out from nose & mouth of terminal patients. But due to discipline, devotion and enthusiasm of each & every staff and cooperation from all concern we could manage many a crisis with ease.

Work in Subodh Mitra Cancer Hospital was carried on with the help of oncology specialist but gradually the researches were conducted independently. Its positivities were associated with modern procedure of treatment. But unfortunately there were commercial problems due to entry of lesser number of patients under conventional treatment resulting loss in revenue. Psorinum was associated with palliative management to form a genuine medical platform alternative to Chemotherapy and Radiotherapy. Later on practically a new platform was formed with non-oncologists instead of oncologists and naturally this gave birth to further internal problems. In spite of explaining to everybody that our aim is to provide relief to cancer patients and not to refuse any scientific treatment, a group of doctors smacked danger in their career and we were accused of insulting modern oncology science and destructing the glorious future of medical science. Notwithstanding the cooperation of many outstanding persons like Dr. Ashok Bose, Mr. Pannalal Guha Thakurata, Hospital Secretary, Mr. Sudin Babu, Dipubabu and others in Subodh Mitra Cancer Hospital, we could not avoid conflict among doctors. Situation gave only two alternatives – either to engage in perpetual internecine war or to leave the institution. I preferred the latter one. Our motto was to include everyone in this noble work and not to exclude anybody. A wide platform was formed including general physicians interested in Psorinum therapy but gradually it led to the rise of an unhealthy atmosphere. In order to avoid that eventuality I decided to detach from Subodh Mitra Cancer Hospital in the middle of the year, 2008.

I am attaching here two letters which would show the importance of the position I held at Subodh Mitra Cancer Hospital.

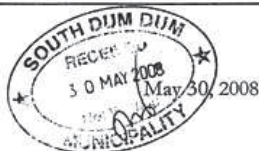


Subodh Mitra Cancer Hospital & Research Centre

1B-175, Salt Lake City Sector - III, Kolkata-700 106

Phone : (033) 2335-5805 / 2335-2515, Fax : (033) 2335-5085

E-mail : subo5531@dataone.in, Website : www.cancerhospital.org




To
The Chairman
South DumDum Municipality
Nagerbagar
DumDum,

Dear Sir,

We are grateful that you have accepted our proposal shortly detection of Breast Cancer and C.A Cervix Jointly with us initially we like to start such Clinic twice in a Month after detection, the diagnosis patients will be referred to Govt, Hospital and to our Hospital.

If you agree to the above proposal we can start working shortly.

Anticipating early decision.


Asoke Bose
Secretary
Subodh Mitra Cancer Hospital &
Research Centre

30/5/08


Dr. Ashim Chatterjee

Coordinator
Subodh Mitra
Cancer Hospital
& Research Centre

30/5/08

Quality Cancer Care at Affordable Cost

The letter addressed to the Chairman of South Dum Dum Municipality, Kolkata,
Mr. Sreehir Bhattacharya

Memo No. SDM/0201/XXIV

Dated: 2nd June, 2008

Office of the Councillors of South Dum Dum Municipality
Nager Bazar, Kolkata-74

To
Dr. Ashim Chatterjee,
Co-ordinator,
Subodh Mitra Cancer Hospital & Research Centre,
IB-175, Salt Lake City,
Sector-III, Kolkata-700 106.

Dear Sir,

With reference to your letter No. Nil dated 30th May, 2008, I like to say that I do agree with your action in starting the Clinic for the detection of **Breast Cancer & C.A. Cervix** jointly in Health Administrative Unit (HAU) Building at Kalindi.

Your best co-operation is solicited.

Thanking you,



Yours faithfully,

Sreehir Bhattacharya
(Sreehir Bhattacharya)
Chairman

The letter addressed to me from the Chairman of South Dum Dum Municipality,
Kolkata, Mr. Sreehir Bhattacharya

Move Towards Oncology Forum

PHASE I:

As I have already mentioned, under the guidance of The School of Tropical Medicine my paper was presented in the Rajasthan session of Indian Science Congress. With this accomplishment one of my missions ended. In my sub-conscious mind I had always dreamt of doing something fruitful for the entire humanity. A nucleus got its shape on which I was working on for past 14 years. After I came back from Rajasthan, I divided my work under two broad sections:

Firstly, it was very important to obtain the drug's Pharmacokinetics and Pharmacodynamics. I had already gained the primary knowledge necessary from the Indian Association for the Cultivation of Science and then from the School of Tropical Medicine. But I knew that it was going to be very difficult and would involve huge amount of time as well as money and it would not be fruitful either. Whether we will receive any government aid in this regard was also a question. Although I had acquired the knowledge as to how much infrastructure, manpower, equipments etc. would be required to combat the situation, I knew, I lacked resources to support the situation. Moreover, the government at some point of time wanted to take over the project. If I had involved the government into this, the duration of my work could have extended by a few years then. Also I had this perception that most of my work had already been documented. But soon I realised that as much as I had thought that my work had been accepted by the medical community, the situation was just the reverse. We also have to keep in mind that Oncology and Onco-science are two completely different subjects. I was sure that my drug had left a positive impact and I could freely use it because of its non-toxicity. I had the training how to convince people and get the work out of them in opposing situations. But if I try to work on the drug's Pharmacokinetics and Pharmacodynamics, I would have to do it staying within certain restrictions. This was not acceptable to me. I thought if I could prove its effectiveness it would become much easier to obtain the drug's Pharmacokinetics and Pharmacodynamics.

The co-operation from the scientific community was also a concern for me. It was a challenge for me to learn the basics from the onco-scientists and eventually make them work with me. Though it was very tough, I had faced similar situations in the Indian Association for the Cultivation of Science. Under the supervision of Dr. Manju Ray, I had learnt the subjects like Advanced Chemistry, Biology and Biological Chemistry there. I had an all-directional study in the field of Cancer research under the supervision of Dr. Manju Ray and Dr. Shelly Bhattacharya. Both of them were core scientists. A world beyond science never existed for them. Initially they were not being able to understand what I was actually looking for.

Secondly, it was very important for me to know what is the definition of a drug in true sense and how it is actually prepared? What are the uses of Mice? How to protect and preserve them? How and what to feed them? How to administer the trial drug? How does cancer evolve? How to infect the Mice with cancer? What is HeLa-Heff? What is LD-50? What equipments are required to perform certain tests and how to utilize the available manpower? How to write a scientific paper? What is an index-journal? And many more questions. All these things I

learnt during my long association with the Indian Association for the Cultivation of Science and the School of Tropical Medicine.

After gaining adequate knowledge I realised that the chief source of my research drug is Mite and this Mite is of a different kind, called Scabies mite (*Sarcoptes scabiei*), which is a parasite and lives on the host's (human) skin and burrows into it. In order to attain a deeper understanding on the entire subject the requirement to work in tandem with the Entomology department rose to heights, and I got proper exposure to this Entomology department in the School of Tropical Medicine. My target then was somehow to enter the Entomology department and how I had done that have already been stated hereinbefore. After coming back from the Rajasthan Session of Science Congress I sat in discussion with some prominent personalities of that time in order to create a blue-print of our future steps to be taken. The suggestion that poured in was encouraging i.e. to research more on Mite. Prof. P. S. Chatterjee, Asstt. Prof. Dr. P. K. Kundu, Prof. Hati, Prof. Brahmachari and Prof. Anup Majumdar advised me to work at the School of Tropical Medicine since I was already researching on Mite in the Entomology department of the institution. Prof. Tandon, my another well-wisher, used to tell me that he had done enough research on Mite. He became very excited and approved the suggestion. He further advised me to mentally prepare myself for the hard time that I might have to face in future. In the meantime, Prof. R. S. Bhakta advised me to join back the Indian Association for the Cultivation of Science. His justification was that since my scientific preparations took place in the Indian Association for the Cultivation of Science, it was the appropriate time for me to go back there. Had they not accepted me, my paper would never have been published under the banner of the School of Tropical Medicine. In fact the most ideal situation would have been to work in collaboration with the Indian Association for the Cultivation of Science and the School of Tropical Medicine. He further suggested to me to consider a few government projects. Again clinical trials were not very important in this case since my trials on human were quite successful. I needed to work more on mice, cell-line etc. This information I conveyed to Prof. Subir Dutta. Although it was a good gesture, however, I felt egoistic. In my years of association with the Indian Association for the Cultivation of Science I became quite fond of it. I used to love its library, the pond where I used to spend hours sitting by its side and lost in thoughts on miles and miles for me to go.

However, just after ten days Dr. Hiranmay Mukherjee told me that there was something still left to be done, that is, to convince the Oncology Forum and gain their approval for my research work. He further stated that whenever he met Oncologists, they used to mock at me and also at the people associated with me. Prof. Manju Dutta Choudhury, then Head of the Haematology department in the School of Tropical Medicine, who was again one of my well-wishers, also said the same thing to me. Since she used to praise me and my work, people used to mock at her as well.

Dr. Mukherjee one day informed me that two renowned oncologists were challenging my hard work of years after years, stating that it was a sin on my part to present a lie misutilizing the prestigious platforms like the Indian Science Congress and the School of Tropical Medicine. They charged me by saying that they would never agree that cancer had completely regressed. Moreover, the School of Tropical Medicine should not have published

my paper in the abstract of their journal in the Indian Science Congress, according to them. They threatened to officially protest against me on every possible platform. Later on at a meeting conducted in the School of Tropical Medicine the members agreed to the fact that the two oncologists I was speaking about were not absolutely incorrect. As I discussed this with Prof. Subir Dutta, he simply said, "Now since the paper has already been published, nobody can do anything about it."

By this time Dr. Hiranmay Mukherjee and I had already planned to contain the Oncology Forum and make them join us. Dr. Mukherjee said that this would be very difficult, since my target was to work with a group consisting of core oncologists. He said: ***You are the head of the subject matter and you have to continue with your work whatever the situation be. You also need to learn certain things staying amidst them. They would be your competitors as well as your colleagues. Although our objective to treat cancer patients is the same, nevertheless, the oncologists are not scientists. It would be very difficult for them to accept what you are proposing since they have come so far to a point after many many years of untiring effort, and then they would come to know one fine morning that a person has been trying to bring down the structure they have built over so many years. They would definitely defend themselves and protest.*** To this I politely submitted that I was not trying to bring down the structure but was trying to enrich it with certain new developments that I had come across and add to its value. Dr. Mukherjee said: ***It is not as it seemed to you. There were two ways in front of you i.e., either create a new platform with physicians from other branches, which was advised by many people around me, or get into a direct conflict with Oncology Forum.*** I replied by saying that if I would prefer to get into a direct fight and good friendship at the same time, then how would that be. Dr. Mukherjee had nothing to say but smiled.

Dr. Mukherjee and I decided that we would approach the Oncology Forum with folded hands and present the facts that we had. At that time Prof. Prabir Sur was the Secretary of the Forum and Prof. Subir Ganguly was the Chairman and they were also respectively the editor and the chairman of JIMA (Journal of the Indian Medical Association), IMA's manifesto and medical journal. Prof. Prabir Sur was the supporter of the then ruling party in the state and Prof. Subir Ganguly had different political ideologies. However, both of them were good friends.

Dr. Hiranmay Mukherjee was friendly with Prof. Prabir Sur and introduced me to him. Prof. Sur at the beginning refused to help stating that everyone in the Oncology Forum was very displeased with me. If we would have approached him earlier he would have advised me that it was not the correct time to go to the Indian Science Congress. Most of the members of the Oncology Forum did not know me and at that time they were avoiding a direct confrontation with Dr. Saroj Gupta's group who were the Oncology Forum's opponent party. But Prof. Sur and Dr. Mukherjee did not know that I had already taken Dr. Gupta in confidence. Prof. Sur said that Prof. Subir Ganguly would never approve of this. Most of the oncologists would follow him and felt very irritated over the entire episode. Ultimately Prof. Prabir Sur told me that he had gone through a few cases that I had treated and assured me of all help. He also pointed out my mistake in not informing Dr. Gauripada Dutta, the famous gynaecologist and the then MLA and Health Committee Chairman, State Legislative Assembly, about my

presentation at the Indian Science Congress session. Prof. Sur said that if he had known he would have definitely stopped me, since it was not the correct time for me to open up.

Later Dr. Mukherjee told me that he was not in good terms with Prof. Ganguly and would not be able to help me in this regard. One day, Dr. Parthojit Banerjee said that he and Prof. Ganguly are good friends and he arranged a meeting for me. He then introduced me to Prof. Subir Ganguly. I was absolutely mesmerised by the presence and behaviour of Prof. Subir Ganguly. He agreed to help me with everything I would need for my research. He asked me to be in constant contact with Prof. Prabir Sur who would be updating him about the progress of my research at every step.

Soon I got involved in the research work with Prof. Subir Ganguly. At that time I was working with many poor patients. One day I took him with me to visit one of my patients. The patient was the wife of a pen-seller (hawker), only 30 year old, and suffering from liver cancer, jaundice and ascitis. She also had a 6 year old son who was looking at his mother as we went and at the same time playing around with 10-12 pens. Prof. Ganguly went emotional. He was carrying a Parker pen in his pocket which he gave to the little boy and the boy took it with a glee. Then we returned back. The very next day when I visited Prof. Ganguly in the hospital, then too he was very emotional. He said: ***Let us find a new horizon. Let us think about the issue in a whole new perspective and without any trivial ego. We must work together to arrive at a lasting solution.***

From the very beginning of my career I have never been an advocate of conventional treatment method for cancer, and if my works are analysed one would realise that I had a far more clear perception of what I wanted to do and how. I had already thought that my choice of the medicine combating cancer would be such that it would not only be one-point solution but also would be easily accessible to. I knew that my path is going to be very difficult, still I have to overcome all odds and emerge undefeated. My aim at first was to know the cancer pathology, and even primarily what is pathology. In my journey I came in close contact with many famous personalities working in the field of cancer and many of them had already left their mark in the history of treatment of cancer. One such of them is Prof. Subir Dutta who was then the Dean of Calcutta University's Medical Faculty, and also the chairman of Pathology Forum. It was my own responsibility to check whether my treatment methods were effective in the treatment of cancer or not. This was a very hard time of my life.

PHASE II:

Even 25 years ago the detection methods were not as advanced as now. I received tremendous support from the Pathology Forum. On the other hand the Oncology Forum, like an enemy, always stood against me. They kept on stressing and objecting on four points – Firstly, how come a man without any medical background represent Bengal and the prestigious School of Tropical Medicine at the Science Congress Session? Secondly, why was the paper presented by me at the Science Congress Session published under the banner of the most prestigious research institute, the School of Tropical Medicine? Thirdly, the member of the Program Advisory Committee of the Directorate of Science and Technology was at that time also the Director of the School of Tropical Medicine, Prof. A. K. Hati. Lastly, the

paper was titled 'The Treatment of Cancer – a step forward' showed that without undergoing Radiotherapy and Chemotherapy, lung, liver, gall-bladder, stomach, pancreas and oral cancer had totally regressed. How it can be?

My aim was to break the prevailing structure of cancer treatment and rebuild it. I had sufficient proof in hand to start a debate, but I did not do so. Here, however, the experience I had gained during the 1970s helped me a lot i.e. "Not enemies, make friends". I then clarified: ***I am now a physician. I have come across many findings and realisations in my journey so far. We are the people who can try to make this world cancer-free.*** I asked them: ***When we are working towards the same goal, even though our paths differ, why there should be any such enmity amongst us? And if you feel that I am going wrong somewhere, please correct me and also help me by showing me the correct way.*** After the years of unending hard work I have learnt a lot and my motto is to make this world a better place to live for the entire human community.

Through this entire controversy I gained attention of a few famous personalities like the then chairman and president of Indian Medical Society, and then member of Oncology Forum Prof. Subir Ganguly. Fortunately Prof. Subir Ganguly became very friendly with me and for many days stood as my pillar of support.

When I got engaged in work with Prof. Subir Ganguly I had to face protests from the junior oncology students as well. Milan (Minu) Dutta, mother of one of the students, at that time was suffering from cancer at the head of pancreas. Dr. Gautam Mukherjee (surgeon) had already performed a by-pass procedure on her. No oncologists wanted to take her case. Prof. Subir Ganguly and my friends from the School of Tropical Medicine warned me not to take her case as well. But Dr. Sharadwat Mukhopadhyay, MD, then a student of Radiotherapy in the Medical College, and his friend, Dr. Animesh Dutta, son of said Milan (Minu) Dutta, challenged me to get her condition improved, if at all. Still I gave my humble efforts for treating Milan (Minu) Dutta. Fortunately the patient's condition improved within a short span of time. Both Dr. Gautam Mukherjee and Prof. Subir Ganguly were excited and overjoyed. Milan (Minu) Dutta till now is alive and is spending a healthy life. This particular case opened the doors of the Radiotherapy department of the Medical College, Kolkata for me. In this context I would also like to mention that after 1.5 years of treating Milan (Minu) Dutta questions started rising as to whether Milan (Minu) Dutta had Cancer at all or not. For near about three months Milan (Minu) Dutta had also stopped taking her medicines, but again she started falling sick. I again treated her and made her stand back on her feet. After this incident no other questions rose concerning efficacy of my Psorinum therapy.

Reports and supporting documents of Milan (Minu) Dutta.

RNH

Dr. Gautam Mukhopadhyay

M. B. S. S. (Cal) M. S. (Bombay)
(Gold Medallist)

CONSULTANT SURGICAL ONCOLOGIST

Sri Aurobindo Seva Kendra (EEDF)

Ruby General Hospital

(Ex Surgeon, Tata Memorial Hospital, Bombay)

Residence :

Flat-2, 71/A, Sultan Alam Road
Lake Gardens, Calcutta 700 033

Tel : 473-9819

OPERATIVE NOTES

Name : Mrs Milan Dutta.

Date: 11/12/96.....

Procedure : Exploratory lapotomy for malignant obstructive jaundice

Chief Surgeon : Dr. Gautam Mukhopadhyay

Asst Surgeon : DR Soumalk Sarkar / DR S. Mukhopadhyay

Anaesthetist : DR Tapan Bose

Physician : DR Kishore Nandy

Time : 2 1/2 hrs

Blood Loss : minimal

Blood Replaced :

Findings And

Technique :

Chevron's incision

No liver metastasis or ascitis or peritoneal nodules

6X5 cm hard lobulated mass head of pancreas
involving medial duodenal wall. / growth extending
to body pancreas / tail free (NON-RESECTABLE)

Triple bypass done + choledoch jejunostomy / gastro-
jejunostomy / jejunojejunostomy by usual technique.

Procedure technically ~~satisfactory~~ satisfactory.

PLAN :

PALLIATION.

Path

Gautam Mukhopadhyay
Signature

NOCLSA
ADMINISTRATIVE CENTER
ALFRED ST. '50
NEW YORK, N.Y.
NEW YORK, N.Y.

Enrique S. Schmitt, P.O. Box 607, Telephone: 334-2941, 334-8560, 359-8396, Fax: (91)/33-3344634

Reprinted on Monday, December 2, 1991.

DEPARTMENT OF C.T.SCAN

Oral and I.V. contrast enhanced CT scan of upper abdomen done by taking 10mm. thin contiguous slices from domes of diaphragm to lower poles of kidneys with further evaluation of porta hepatis and head pancreas region by 5mm. thin slices.

BONES
Bones under review are normal showing intact cortical outline.
Paravertebral & parietal muscles are normal.
Lower pleural spaces are clear on both sides.

Liver is normal in size, shape, outline and shows homogenous density with no focal SOL or any deposit. Only main bile duct are seen prominent. Porta hepatis region is normal.

PANCREAS

SPLEEN

Control . . . 2 . . .

Suraksha

Suraksha Diagnostic & Eye Research (Pvt.) Ltd.

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Page No. 2 of 3

Document No. 1070513 Printed on Monday, December 2, 1996

ADRENAL GLAND

Adrenal gland of both sides are normal in position, size, shape, density and contrast enhancement.

KIDNEYS

KIDNEYS
Both kidneys are normal in position, shape, size, contour, density and contrast enhancement. Prompt excretion of contrast is seen from both sides. Both pelvicalyceal system are normal. No calculus or hydronephrosis is seen.

BOWEL - MESENTERY AND PERITONEAL CAVITY

BOWEL : MESENTERY AND PERITONEAL CAVITY
oral contrast distended bowel loops are normal.
No sizeable solid / cystic SOL is seen in mesentery.
No calcification is seen. ~~pancreatic duct is not dilated.~~
No free fluid . peritoneal thickening or nodularity is seen in
peritoneal cavity.

RETROPERONEUM

RETROPERITONEUM
Aorta and IVC are normal. No retroperitoneal lymphadenopathy or any
SOL is seen.

Contd...3...

Suraksha

Suraksha Diagnostic & Eye Research (Pvt.) Ltd.

18 CIT Road, Scheme VII, Calcutta-700014, India. Tel: (91) 93 4344634

Report No. L020513 Page No. 3 of 3 Printed on Monday, December 2, 1996.


IMPRESSION CT scan of upper abdomen reveals.

- 1) Early biliary tree dilatation with dilatation of main bile ducts and common bile duct, the lower end showing regular tapering adjoining head pancreas.
- 2) Pancreatic duct is seen prominent and shows early dilatation.
- 3) Inferior part of head pancreas and adjoining medial part of uncinate process shows disproportionate focal enlargement however no altered attenuation or enhancement is seen and surrounding fat planes are maintained.
- 4) No sizeable deposits evident in liver or coeliac, mesenteric lymph nodes.

Possibilities - ? neoplasm of lower end of CBD.

- ? neoplasm of head / uncinate process of pancreas.

With compliments for kind referral.


DR. GAUTAM GHOSH M.D.
Radiologist

02.12.96

Prepared By : KRR
Printed By : SMB

AUGUST SPECIALITY HOSPITAL

H-3, ALIPORE ROAD, CALCUTTA - 700 027 • PHONE 4762995, 4762996 • FAX NO. 4762997

Report No. _____	Date _____
Ref. No. _____	Reg. No. _____
Name _____	Bed No. _____
Doctor _____	Class _____
Age _____	Sex _____

Referred by : _____

INDICATION : _____

History : Inj. IV. Calapose 10mg
Inj. for twin 10mg
Inj. for opant 30mg

PHYSICAL : No signs of small papilla
Findings : _____

ERCP : PANCREATOGRAM : Pancreatic duct is dilated and tortuous in the body and tail region with narrowing in the head region.

CHOLANGIOGRAM : CBD is dilated with narrowing near the ampullary end.
No stone seen.

Remarks : Obstructive Jaundice-stricture lower end of pancreatic duct and CBD. ? cholangio CA.
? Pancreatic head SOL with bile duct invasion.
Suggest : C.T scan of abdomen. (upper)

S. Panigrahi
Consultant In Charge

ERCP =
ref

Dr. Jaydip Biswas

MBBS, MS, DMRT, FAIS, FICS
Consultant Surgeon & Oncologist

Date 8/8/03

Ref.

Mrs. Milan Dutta
51yrs F

1. Case of
Obstructive jaundice
(? malignant)

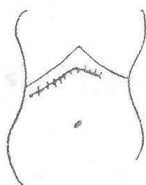
Triple bypass done in 1996.

is a case of
non-resectable
pancreatic growth.

Pl. is doing well
since liver test
May 2003

Developed weakness
jaundice and loss of appetite for 2 months
Badder / day.
Bowel ↓

On exam



G.I. good G.S. Anaemia +
neck nodes - NR.

Chest - clear clinically

Abdomen - no lump
no detectable
free fluid clinically

Lab.

Blood for Hb).

TLC, DLC & platelet
count.

Blood for liver
function test

Blood for urea / Sugar
(F.P.)

ET scan abdomen

U.S.G. whole
abdomen

Residence : EE-174 A/2 (Near 10 No. Tank Petrol Pump), Salt Lake City, Kolkata - 700 091
Phone : 321-9878, Mobile : 98300 26696, E-mail : drjaydip_biswas@yahoo.com

3. Cal Fersdale - ca
 . Cal Farizyn full 2hbs
 2 2. 1.
 Cal Belybin 1m

8/8/07

EKO DIAGNOSTIC

A Unit of Eko Diagnostic Pvt. Ltd.
54, Jawaharlal Nehru Road,
Kolkata-700 071
Ph. : 282-9246/8105/8106/8109
Fax No. : 282-8098
E-mail : ekoxray@satyam.net.in



DIVISION OF IMAGING

MRS MILAN DUTTA

51 YEARS

09.08.2003

DR JAYDIP BISWAS

C T SCAN OF UPPER ABDOMEN

HISTORY

? Weakness, pruritis. Follow up case of non-resectable growth of pancreatic head with triple bypass done.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced C T scan of the upper abdomen done with 5 mm. & 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no significant abnormality.

Liver is normal in size, shape, position, outline and density. Mild changes of pneumobilia are seen in a few intrahepatic biliary radicles. The intrahepatic biliary radicles are not dilated. No focal lesion is detected.

Gall bladder is not clearly visualized and appears to be filled with oral contrast and little air (history of cholecysto-jejunostomy)

Common bile duct appears normal with evidence of air within its lumen.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Contd....2..

EKO DIAGNOSTIC

A Unit of Eko Diagnostic Pvt. Ltd.
54, Jawaharlal Nehru Road,
Kolkata-700 071
Ph. : 282-9246/8105/8106/8109
Fax No. : 282-8098
E-mail : ekoxray@satyam.net.in



DIVISION OF IMAGING

2

MRS MILAN DUTTA

(CT OF UPPER ABDOMEN)

09.08.2003

Both suprarenal glands reveal normal morphology and density. They are normal in size. No evidence of nodularity or SOL is seen in the suprarenal glands.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal. Both upper ureters show normal course and calibre.

Aorta and IVC are normal. Left para-aortic lymph nodes are present. No free fluid is seen in the peritoneum.

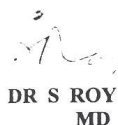
Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

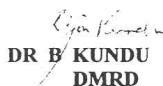
IMPRESSION

C T features suggest evidence of pneumobilia in a few intrahepatic biliary radicles and in the common bile duct. The gall bladder is not well visualized. A few small left para-aortic lymph nodes are suggested. No other detectable abnormality is noted.

Suggested clinico-pathological correlation and other investigations for further evaluation and confirmation.


DR S K SHARMA
MD


DR S ROY
MD


DR B KUNDU
DMRD

DR S KUNDU
MD

MILAN DUTTA**64 YRS****30.05.2016****DR. OF MCH****4071/018****CT SCAN OF WHOLE ABDOMEN****HISTORY**

Post-operative follow up patient of nonresetable pancreatic growth – had triple bypass doing well now.

TECHNIQUE

Plain, oral and I.V. (non –ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no obvious abnormality.

Liver is mild enlarged. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Porta hepatis appears normal.

Gall bladder is not visualized.

Common bile duct is not dilated.

Pancreas : Visualized pancreas is normal.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

-2-

MILAN DUTTA**64 YRS****30.05.2016**

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Pelvic organs are normal.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

IMPRESSION


CT Scan of abdomen is within normal limit.

Pancreas as visualized is morphologically normal.

Suggested clinical correlation and further investigations if clinically indicated.

DR.D.SHARMA
MD

DR T.K.DHAR
MD (Radiodiagnosis)


DR.ANUP SADHU
DMRD, MD

2

MILAN DUTTA**64 YRS****30.05.2016****DR. OF MCH****4073/018****CT SCAN OF THE CHEST****HISTORY**

Follow up patient of Ca. head of pancreas.

TECHNIQUE

Plain & I.V. (non-ionic) contrast enhanced C T scan of the chest done with 5 mm. and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the chest in supine position and in frontal projection shows no detectable abnormality.

Bones under review shows no detectable abnormality. Parietal muscles appear normal. No sizeable mass is seen in the axillae.

No evidence of pleural thickening or pleural effusion is seen.

Great vessels of the mediastinum including ascending aorta, arch of aorta and its branches, descending aorta, M.P.A. and its branches, S.V.C. and its tributaries appear normal. Cardiac outline is normal. Trachea and its bifurcation are normal. No sizeable mass is detected in the mediastinum.

No focal lesion is detected in the lung parenchyma. Broncho-vascular markings are normal.

IMPRESSION

CT Scan of chest is within normal limit.

Suggested clinical correlation and further investigations if clinically indicated.

DR.D.SHARMA
MD**DR T.K.DHAR**
MD (Radiodiagnosis)**DR.ANUP SADHU**
DMRD, MD.

Hs. Milan Dutta
 51 yrs F.

F.H. Case of
 obstructive jaundice
 triple bypass Apr. 1996
 in a non resectable pancreatic
 growth.

Patient is presently
 asymptomatic

CT Thorax } No evidence
 CT Abdomen }

Clinically
 No measurable
 disease.

Adh.
 Lab. values
 3/10
 1/10 1/10
 X 1/10

Cap. Econom
 1 Cap 1/10
 X 1/10

Planned for Jan 1997.
 Info. Los if symptomatic

Next check up
 after 3 months

24/6/2006



Milan Dutta with Prof. Jaydeep Biswas and Dr. Asim Chatterjee

I used to discuss with Prof. R. N. Brahmachari on various subjects and often seek his advice. With respect to my research work, he once said: *The work you are involved in can be classified under two broad sections: Firstly, the drug, the materials used to prepare it, methodology of its preparations, its effect on the human body; and the procedure of administration of the drug, its balanced duration, do's and don'ts, post-treatment requirements or, in other words, the Pharmacokinetics and Pharmaco-dynamics of the drug along with its therapeutic value. Secondly, whether the disease is actually Cancer, as reported by the pathologists, or not. You have been able to clearly distinguish that as well. You have also understood what a Forum is. Prof. Subir Ganguly has already tested you. And you have proved over and above that the patients you have treated were actually suffering from cancer. Your work also has been acknowledged at the Annual Conference (IAPM), organised under the supervision of Prof. Subir Dutta, under the heading "Promising result of a traditional Drug 'Psorinum' on carcinoma of rectum- A case study" in the West Bengal Chapter – Abstracts.*

The paper was published under the guidance of Prof. Dutta himself and the School of Tropical Medicine. Prof. Dutta being the then active member of the Pathology Forum and also the Head of Dept. Of Pathology in the University College of Medicine, in association with Dr. A. K. Hati, the then institutional head of the School of Tropical Medicine and also the Head of Department of the Entomology, before Dr. Hiranmoy Mukherjee, and a few of my fellow colleagues like Dr. P.K. Kundu, Dr. P.K. Bhattacharya, Dr. Parthojit Banerjee, Prof. R.S. Bhakta and Dr. Hiranmoy Mukherjee. The Pathology Forum in one of their conferences in Kolkata acknowledged that the rectum cancer treated by me had actually been cancer, and that conference was attended by many renowned doctors of Kolkata. This implied that the West Bengal Chapter on Pathology also acknowledged the success of my works. But that the disease has completely cured can be certified only by the Oncology Forum. As I mentioned earlier, their approval was difficult to obtain. I was not openly associated with the Oncology Forum, and from the very beginning it used to work under the twin leadership of Prof. Subir Ganguly and Prof. Prabir Sur. Both of them were the students of Prof. R. N. Brahmachari but they were not much in touch with him. Prof. R. N. Brahmachari advised me that it would be better if I could confine my work to clinical trials and to the Pharmaco-kinetics and Pharmaco-dynamics component of the drug. The reference can be found in my twelve-year long association with the School of Tropical Medicine the details of which I have provided in this book.

PHASE III:

My next objective was to publish some papers in collaboration with at least one active member of the Oncology Forum. That person would eventually become my referee and guide. I knew that this would be very tough because the entire responsibility would rest on his shoulders. But somehow I managed to take Prof. Ganguly on board, since he was an open-minded person. He got associated with me and we together wrote a few papers under the banner of the NGO, New Resource. Later on under the joint venture of New Resource and Oncolink, a few more papers were published.

After the success of Minu Dutta's case more oncologists got associated with us, and together we submitted an Abstract at the prestigious 6th International Congress on Oral Cancer, 15th – 18th of February, 1999, under the banner of Oncolink. Subsequently the entire paper was published and later on was presented by us in the seminar. With this we were able to draw attention of the international scientific world. The Committee received ample support from the Ministry of Health & Family Welfare, Ministry of External Affairs, Ministry of Home Affairs, Directorate of Science & Technology, ICMR, DRDO and INSA at the national level. On the international front the organisations that lended support were UICC (Switzerland), NIH (USA), NIDR (USA) and AUSAID (Australia). During that time I was also attending Mr. Brojen Das who was a world famous swimmer and was suffering from lung cancer. He was under my treatment for near about a year then. His son-in-law, the famous scientist Dr. Sanjay Paul, also got associated with me and we worked together for some time. He accompanied me to Delhi to attend the seminar, 6th International Congress on Oral Cancer. At a later stage he moved to BHU (Benaras Hindu University). However, he had by that time gained interest in the study of alternative medicine and kept practising it. He also has a few international publications on alternative medicine by his name.

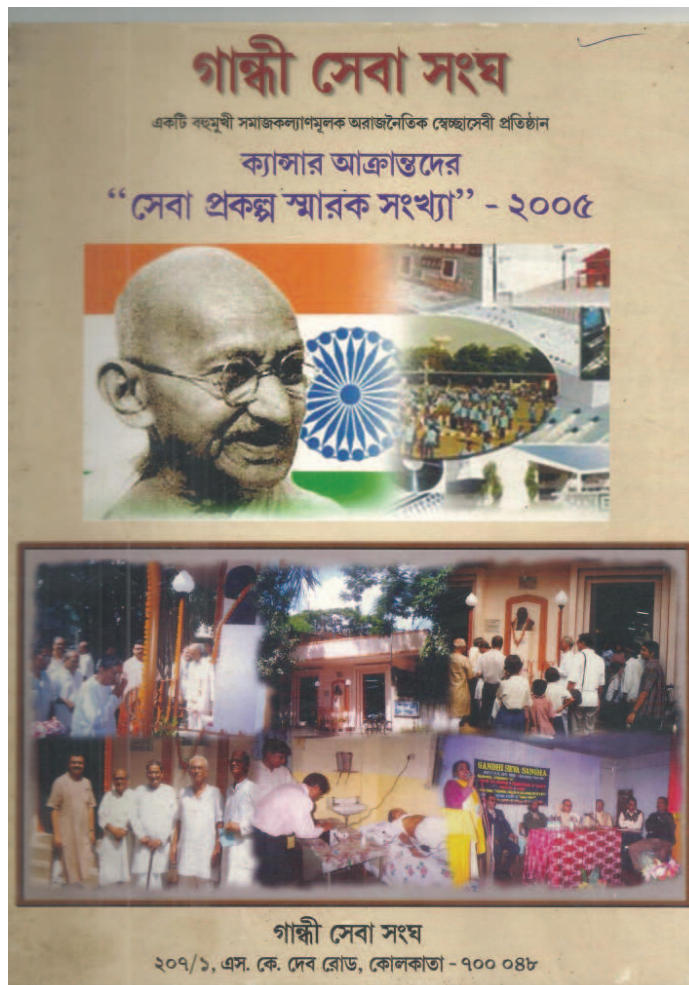
This was the time I got introduced to Major General Dr. A. K. Varma in Delhi. I have already discussed about our association and also the warmth we shared towards each other. He knew that some of my works had been published by then in the prestigious international journal named Lacet Oncology, and they had reviewed it and opined my therapy as truly effective.

MY BRIEF ASSOCIATION WITH GANDHI SEVA SANGHA, Sreebhumi, Kolkata

After resigning from Subodh Mitra Cancer Hospital I joined Gandhi Seva Sangha (voluntary charitable organisation used to come to Kolkata from treatment of cancer, but their family guest houses at huge cost. I proposed to the authorities of Gandhi Seva Sangha to arrange a place of stay for such Cancer patients and their accompanying family member(s) at Gandhi Seva Sangha itself. They agreed to my proposal and constructed the first floor of Gandhi Seva Sangha for the purpose. On a piece of 1.5 bighas of land lying with Gandhi Seva Sangha Research Institute, but the senior member of the Gandhi Seva Sangha were perhaps not interested in my proposal. Instead they offered me to build a General Hospital, but I stood firm at having Cancer wing constructed with the facility of radio modern Cancer management system. Hence, some disputes sprang up. I ultimately left Gandhi Seva Sangha's core committee. During my tenure at both Subodh Mitra Cancer Hospital and Gandhi Seva Sangha I was also continuing with the NGO, 'Combat Cancer seminar was organised at the University Institute Hall, Kolkata, and I participated in the Seminar.



Gandhi Seva Sangha



সভাপতির প্রতিবেদন —

মাননীয় প্রধান অতিথি ব্রীড়া ও পরিবহন মন্ত্রী শ্রী সুভাষ চক্রবর্তী মহোদয়, মাননীয় বিশিষ্ট অতিথি আবগারী শুল্ক মন্ত্রী প্রবোধ সিংহ মহাশয়, বিশিষ্ট অতিথি মাননীয় প্রাক্তন কেন্দ্রীয় মন্ত্রী শ্রী তপন সিকদার, সুবোধ মিত্র ক্যানসার হাসপাতালের মাননীয় চেয়ারপার্সন শ্রীমতি রমলা চক্রবর্তী, দক্ষিণ দমদম পৌরসভার সম্মানীয় চেয়ারম্যান শ্রী শ্রীহীর ভট্টাচার্য এবং মধ্যে উপবিষ্ট আমন্ত্রিত বিশিষ্ট অতিথিগণ এবং সমবেত সুখী মন্ডলী।

আজ এই শুভলগ্নে আপনারা গান্ধী সেবা সংঘ ‘সেবানিবাস’ এর কল্যাণকর উদ্বোধন অনুষ্ঠানে উপস্থিত হয়ে আমাদের যে আন্তরিকতার পরিচয় দিয়েছেন, তাতে আমার হৃদয় অব্যক্ত আনন্দে পূর্ণ হয়ে উঠেছে। এই এলাকার সবচেয়ে প্রাচীন ঐতিহ্যময় গান্ধী সেবা সংঘ পরিচালকদের তরফ থেকে আমি আপনাদিগকে আন্তরিক অভিবাদন জ্ঞাপন করছি। অভিবাদন জানাচ্ছি নবনির্মিত ধাত্রীরূপী সেবানিবাসের পক্ষ থেকে এবং অভিনন্দন জানাচ্ছি এলাকার সহস্র সহস্র ভাই বোনদের পক্ষ থেকে।

আমি গর্বিত যে আমি গান্ধী সেবা সংঘ পরিবারভুক্ত একজন সাধারণ সেবক হিসাবে পরিসেবা করার সুযোগ পেয়েছি, যে সংঘ পরিবার এই এলাকার অসহায় দরিদ্র পীড়িত রোগীদের তিনজন প্রবীণ অভিজ্ঞ চিকিৎসকের সাহায্যে অবিরাম সেবা করে চলেছেন। আমি বলতে গর্ব বোধকরি যে আমরা গান্ধী সেবা সংঘের মাধ্যমে এলাকার অক্ষম, নিঃস্ব রোগাক্রান্তদের নিরাময় করার উদ্দেশ্যে দাতব্য চিকিৎসালয় স্থাপন করতে পেরেছি।

উপরন্তু, স্বনির্ভরতা প্রশিক্ষণ কেন্দ্র সংস্থাপন, একটি গ্রন্থাগার পরিচালনা, সংস্কৃতি মঞ্চ প্রতিষ্ঠা, শিশুবিকাশ কেন্দ্র প্রবর্তন, ইত্যাদির বিভিন্ন মাসুলিক কাজের মাধ্যমে জন কল্যাণমূলক কাজে নিজেদের নিযুক্ত করতে পেরেছি। এই বিভিন্ন পরিসেবার যে অনন্ত অভিজ্ঞতা অর্জন করতে পেরেছি তার ফলে এই এলাকার বহু সংখ্যক রোগার্তদের এবং বহিরাগত বিপন্ন দুঃস্থ লোকদের উদ্দেশ্যে আরও বৃহত্তর সেবামূলক কাজ করতে আমাদের উদ্বীপ্ত করেছে। গান্ধী সেবা সংঘ এবং নবজাত এই সেবানিবাস দক্ষিণ পূর্ব কোলকাতায় একটি ঐতিহ্যময় অবিস্মরণীয় প্রতিষ্ঠান। আজ থেকে ষাট বৎসর পূর্বে প্রাচীন কোলকাতার জোড়াসাকো অঞ্চলের (১২ শিব কৃষ্ণ দাঁ লেন, কোলকাতা - ৭) বিখ্যাত ধনাঢ্য দাঁ পরিবারের কৃতী সন্তান প্রয়াত শ্রদ্ধেয় দরিদ্র বন্ধু পুলিন চন্দ্র দাঁ, স্বর্গীয় অনিল চন্দ্র দাঁ ও স্বর্গীয় বঙ্কিমচন্দ্র দাঁ মহোদয়দের আন্তরিক বদান্যতায় প্রাপ্ত দুবিঘা জমির উপরে

১৯৪৬ সালে প্রতিষ্ঠিত হয়েছে গান্ধী সেবা সংঘ। এই প্রতিষ্ঠান গঠনে সহযোগিতা করেছিলেন তদানীন্তন কোলকাতার প্রসিদ্ধ দত্ত পরিবারের উত্তর পুরুষ স্বর্গত দুর্গা চরণ দত্ত ও অনন্তলাল দত্ত। পরবর্তী কালে এই সংস্থায় যোগদান করেছেন প্রয়াত বিশ্বনাথ লাহা, অখীর কুমার বসু, মানিক্যরতন ওহঠাকুরতা, সর্বশ্রী গৌরঙ্গ চন্দ্র বনিক ও শ্রী বাচোয়াত। এই সংস্থায় প্রচুর অর্থ দান করেছেন স্বর্গগত হেমেন্দ্রলাল কুড়ু মহাশয়। ষাট বৎসর অতীতের সেই ক্ষুদ্র অঙ্কুর সম প্রতিষ্ঠানটিকে সম্বলে লালন করে এবং দীর্ঘকালব্যাপী অপরিসীম শ্রম ও প্রযত্নের দ্বারা ধীরে ধীরে প্রয়াগের অক্ষয় বটবৃক্ষের মতো সৃষ্টি হয়েছে আমাদের আদরের প্রতিষ্ঠান গান্ধী সেবা সংঘ, এবং ইহা বর্তমানে বিশাল মহীরুহে পরিণত হয়ে লাভনি রূপ ধারণ করে ইতিহাসে স্থান লাভ করার যোগ্যতা অর্জন করেছে।

আজ এই স্মরণীয় মঙ্গলময় লয়ে বিগতযুগের প্রতিষ্ঠিত গান্ধী সেবা সংঘের নব সংবৃত্ত মৃত্যুঞ্জয়ী সেবা নিবাসের মাস্টলিক উদ্বোধন হচ্ছে। এই সেবানিবাসের প্রাণপুরুষ ডাঃ অসীম চ্যাটার্জী মহাশয়ের অভিনিবিষ্ট উদ্যোগ, সেই সঙ্গে রয়েছে সংঘের সাধারণ সম্পাদক শ্রী গৌতম সাহার একনিষ্ঠ সৃজনশীল সাধনা এবং সংঘ কর্তৃপক্ষের প্রতিভাশীল সদস্যদের ঐকান্তিক প্রচেষ্টা ও আর্থিক অবদান যার ফলে আমাদের এই কল্যানময় কর্মযজ্ঞ সুসম্পন্ন হয়েছে।

এই গৌরবময় পরিকল্পনায় বিশেষভাবে সহযোগিতা করেছেন আমাদের শুভাকাঙ্ক্ষী সুবোধ মিত্র ক্যানসার হাসপাতালের চেয়ারপার্সন মাননীয় শ্রীমতি রমলা চক্রবর্তী মহাশয়া এবং হাসপাতালের খ্যাতনামা চিকিৎসাবিজ্ঞানী প্রমুখগণ।

আমি আশা করি ক্যানসার রোগাক্রান্ত রোগীদের নিরাময় করার জন্য এবং তাদের নিকট আত্মীয়দের এখানে অবস্থান করার উদ্দেশ্যে নির্মিত সংঘসেবানিবাসটি হবে একটি পরম পবিত্র আরোগ্যনিকেতন। পরিশুদ্ধ জ্ঞানময় প্রজ্ঞা দ্বারা আমাদের সকলের অন্ত ইচ্ছাশক্তিকে যদি সম্পূর্ণ রূপে প্রদীপ্ত করতে পারি তাহলে 'সেবা নিবাসকে' এদেশের অন্যতম শ্রেষ্ঠ আরোগ্য নিকেতনে প্রতিষ্ঠিত করতে পারবো, এ বিশ্বাস আমার অন্তরে রয়েছে। আমি আরও আশা করি এখানকার রোগীদের প্রাণপণে সেবা যত্ন করে আমি কৃতকৃতার্থ হতে পারবো।

প্রাচীনকালে উপনিষদের মন্ত্রদ্রষ্টা ঋষিগণ পারস্পরিক কল্যাণের আদর্শ স্মরণ করে মন্ত্র রচনা করেছিলেন — সর্বে সন্তু নিরাময়ং কিংবা দরিদ্র দেবোভব, মুমূর্ষু দেবোভব ইত্যাদি। সেই ঋষিগণ সমাজের সর্বসাধারণের বৃহত্তর কল্যাণের জন্য সকলের নিরাময় প্রার্থনা করেছিলেন। অসহায় দরিদ্র রোগাক্রান্ত ব্যক্তিদের দেবাতজ্ঞানে সেবা করতে শিখিয়েছিলেন। তাঁরা অনুভব করেছিলেন যে প্রতিটি মানুষের কল্যান কামনাই আদর্শ সমাজের লক্ষ্য। এই পবিত্র সেবা ব্রতের মধ্যে আছে

দয়ামৃত। এই দয়া কেবল বাইরে নয়, অন্তরের গভীর অনুভূতিতে পূর্ণ। সেবা ধর্মের প্রকৃত আদর্শ অন্তরের গভীর অনুভূতিতে পূর্ণ। সেবা ধর্মের প্রকৃত আদর্শ অন্তরের গভীর আকৃতিতে এবং মমত্ববোধের সার্থক প্রতিষ্ঠায়।

বীর সন্ন্যাসী স্বামী বিবেকানন্দ উদাত্ত কণ্ঠে ঘোষণা করে গেছেন — “বহুরূপে সম্মুখে তোমার, ছাড়ি কোথা খুঁজিব ঈশ্বর। জীবে প্রেম করে যেই জন, সেইজন সেবিছে ঈশ্বর”।

তিনি প্রগাঢ় বিশ্বাসে উদ্বেল হৃদয়ে বলেছিলেন — “যতদিন ভারতবর্ষে একটি লোক ও বুবুক্ষু থাকিবে ততদিন আমি মুক্তি চাহিনা।”

মহাপ্রভু শ্রীচৈতন্যদেব উপদেশ দিতেন — সেবা ও পরোপাকারই মানব জীবনের স্বার্থকতা। তবে তাতে যেন স্বার্থসন্ধান না থাকে। তিনি উপমা দিয়ে বলেছেন যে বৃক্ষ সকলকে ফল ও ফুল দান করে, আর্ত-ক্লান্তদের স্নিগ্ধ ছায়া দান করে, কিন্তু বৃক্ষ কোন কিছুই প্রত্যাশা করে না। মহাপুরুষদের এই সকল দার্শনিক উপদেশামৃত থেকে আমাদের সেবামূলক শিক্ষালাভ করতে হবে। তবেই আমরা কৃতকার্য হবো।

আজ এই শুভ মুহূর্তে সকলের নিকট বিনীত আবেদন আসুন এই বিশাল কর্মযজ্ঞের সফলতা অর্জনের জন্য দৃঢ় সঙ্কল্প নিয়ে আত্মনিয়োগ করি। সকলের সমবেত মহান্ প্রচেষ্টায় কোন কিছুই অসাধ্য থাকে না। আমরা যেন গান্ধী সেবা সংঘের মাতৃরূপী সেবা নিবাসকে নিষ্ঠাময় আরোগ্য নিকেতন রূপে সাজিয়ে তুলতে পারি অন্তরের নিষ্ঠা ও সহানুভূতির অর্ঘ্য দিয়ে।

আজ এই পবিত্র শুভক্ষণে আপনাদের জ্ঞাপন করি আমার আন্তরিক অভিবাদন এবং ভালবাসা। বিশ্বনিয়ন্তা ঈশ্বরের চরণে আপনাদের সুন্দর স্বাস্থ্য এবং শান্তিময় দীর্ঘায়ু প্রার্থনা করি। জয় হিন্দ।

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মনি চক্রবর্তী
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তৃতীয় বিশ্বে ক্যান্সার চিকিৎসা নতুন করে ভাবার সময় এসেছে

ক্যান্সার এক ভয়ঙ্কর অভিশাপ একবিংশ শতাব্দীর মানুষের কাছে। বিজ্ঞান সাধনার সুফল ও কুফল সামাজিক মানুষ নিঃশর্তে ভোগ করে চলেছে আবহমান কাল ধরে। বিজ্ঞান মানুষের জীবনে স্বচ্ছন্দ্যবিহারের এক সম্ভাবনাময় আশা। অথচ বিজ্ঞানের আশীর্বাদ ক্যান্সার চিকিৎসায় যুগান্তকারী আবিষ্কার সেভাবে চোখে পড়ছে না। পৃথিবীর সর্বত্র প্রাচ্য ও পাশ্চাত্যে এ নিয়ে বহুকাল ধরে গবেষকরা নিরন্তর প্রচেষ্টা চালিয়ে যাচ্ছেন। কিন্তু কোনভাবেই আর দশটা সাধারণ রোগের চিকিৎসার মত কোন জীবনদায়ী ঔষধ আবিষ্কৃত হয়নি।

পৃথিবীর অন্যান্য প্রান্তে সামান্য যতটুকু আবিষ্কার লক্ষ্য করা গেছে সেই পথেই ভারতীয় চিকিৎসা ব্যবস্থার সীমাবদ্ধতার ক্ষেত্র গড়ে উঠেছে। ভারতীয় বিজ্ঞানী / গবেষক / ডাক্তাররা ক্যান্সার চিকিৎসা ক্ষেত্রে সেভাবে কোন উল্লেখযোগ্য অবদান রেখেছেন বলে আমার জানা নেই। একটি চালু কথা আছে যে, যে দেশের সমস্যা সেই দেশের ছেলে / মেয়ে সেই দেশের মাটিতে বসে সেই দেশের উপকরণ নিয়ে কাজ করতে না পারলে সেই দেশের সমস্যার সমাধান হয়না। বাস্তবিক এই চিকিৎসার ক্ষেত্রে ভারতবর্ষের অবস্থা বেশ করুন।

বিশেষত ভারতবর্ষ তৃতীয় বিশ্বের দেশ/এখানে অধিকাংশ মানুষই দারিদ্র সীমার নীচে বসবাস করে থাকেন। এখানে ক্যান্সারের মত ব্যয়সাপেক্ষ চিকিৎসার ন্যূনতম সুযোগ পাওয়াও সম্ভব নয়। মোটামুটিভাবে দেশের ১৪-১৫ শতাংশ মানুষ মাসিক ২৫ - ৩০ হাজার টাকা রোজগার করে থাকেন। বাকী ৮৫-৮৬ শতাংশ মানুষ এই জাতীয় ব্যয়সাপেক্ষ চিকিৎসার কোন সুযোগই লাভ করতে পারেন না। অধিকাংশ মানুষই বিনা চিকিৎসায় অকালে প্রাণ হারান।

আমাদের দুর্ভাবনাটা গরীব মানুষদের নিয়ে। আজকাল গ্রামেও বেশ কিছু মানুষের আর্থিক সঙ্গতি আগের থেকে অনেক উন্নততর হয়েছে। গ্রাম্যজীবনের ছবিটাও বদলে গেছে সময়ের সঙ্গে সঙ্গে। মনে রাখা প্রয়োজন, ক্যান্সার চিকিৎসার সামান্য সুযোগ সুবিধা এবং উন্নততর পরিকাঠামো ও অত্যাধুনিক চিকিৎসা ব্যবস্থা সমস্তটাই কিন্তু শহরকেন্দ্রিক। গ্রামীণ জীবনে কোন পরিকাঠামোই পাওয়া সম্ভব নয়। তার মধ্যে মানুষের অজ্ঞতা, সংস্কার এবং শিক্ষার অভাবের ফলে ক্যান্সার সম্পর্কে কোন ধারণা তাদের নেই। যদিও ক্যান্সার কিচিৎসাতে আজও আমরা সঠিক answer দিতে পারিনি, কিন্তু ক্যান্সার মানেই মৃত্যু নয়, আজ খানিকটা হলেও এ সত্য অনেকটাই পরিষ্কার।

মানুষের এ হেন নানা ধরনের সমস্যার কথা মাথায় রেখে ‘গান্ধী সেবা সংঘ’ মানবকল্যাণে এগিয়ে এসেছে মানুষের কাছাকাছি থাকবার জন্য। ব্যাপারটা একটু পরিষ্কার করে বলা যাক।

মনে করা যাক, যিনি ক্যান্সারে আক্রান্ত হয়ে পরিবারের সকলকে ফেলে বেশ কিছুকাল পরে মারা গেলেন, তিনি এজীবনের সমস্ত জুলা-মুদ্রনা থেকে মুক্তি পেলেন। কিন্তু যারা তাঁর পরিবারের লোকজনেরা রয়ে গেলেন, তাদের প্রকৃত অবস্থাটা কোন্ জায়গায় গিয়ে দাঁড়িয়েছে? শারীরিক, মানসিক ও অর্থনৈতিক - সমস্ত দিক থেকেই আজ তারা পিছিয়ে পড়েছেন। এমনও হয়, যে পরিবারের একমাত্র উপার্জনশীল মানুষটিই ক্যান্সারে আক্রান্ত হয়ে মারা গেলেন। পরিবারটিই তখনই হয়ে গেল।

‘গান্ধী সেবা সংঘ’ এই অসহায় মানুষদের পাশে দাঁড়বার অঙ্গীকার করছে। এখানে নানাভাবেই পুরো বিষয়টিকে ধরবার চেষ্টা করা হয়েছে। যেমন :

১) গরীব ও অল্পবয়সে উপার্জনশীল মানুষটির মৃত্যুর পর তার পরিবারের লোকজনদের মধ্যে কাউকে বিভিন্ন ট্রেনিং-এর শিক্ষাদান ও শিক্ষাশেষে কোথাও যাতে কিছু করে আবার পরিবারটি উঠে দাঁড়াতে পারে তার জন্য সর্বোত্তমভাবে সাহায্য করা।

২) ক্যান্সার সম্পর্কে জনমানসে সচেতনতা বাড়ানোর দায়িত্ব গ্রহণ করা।

৩) গ্রামের গরীব ও অসহায় মানুষদের থাকবার ব্যবস্থা করে দেওয়া, যারা শহরে এসেছেন পরিবারের আক্রান্ত লোকটির চিকিৎসা করাতে এবং যাদের হোটেলে থাকবার আর্থিক সঙ্গতি নেই, কলকাতা শহরে যাদের আত্মীয় নেই অথবা আত্মীয়ের বাড়ীতে থাকা সম্ভব নয়।

৪) আক্রান্ত পরিবারের গরীব মানুষদের অর্থনৈতিক স্বাবলম্বী করে তুলতে নানাভাবে তাদের সাহায্য করা।

৫) ক্যান্সার চিকিৎসা ক্ষেত্রে বিভিন্ন প্রতিষ্ঠানের সঙ্গে যৌথভাবে কাজকর্ম করে যাবার ভাবনা চিন্তা ‘গান্ধী সেবা সংঘ’ গ্রহণ করেছে। শুধু ভাবনা-চিন্তাই নয়, একসঙ্গে কাজ করা ও গরীব মানুষদের সেবাকল্পে এগিয়ে এসেছে। এমনই একটি প্রতিষ্ঠান হল - সুবোধমিত্র ক্যান্সার হাসপাতাল এবং রিসার্চ সেন্টার। এটি ক্যান্সার চিকিৎসা ক্ষেত্রে এখনো পর্যন্ত একটি সেরা প্রতিষ্ঠান।

৬) ক্যান্সার চিকিৎসা ক্ষেত্রে অন্যান্য সম মনোভাবাপন্ন বিভিন্ন প্রতিষ্ঠানকেও আমরা সাদরে আহ্বান জানাচ্ছি। আসুন আমরা হাতে হাত মিলিয়ে একসঙ্গে কাজ করি।

৭) সর্বোপরি বলা যায় যে, ‘গান্ধী সেবা সংঘ’কে সামনে রেখেই আগামীদিনে আমরা

পথ চলতে চাই। তাই এটিকে এক সাথে বলা যেতে পারে Research Model। লক্ষনীয়, ক্যাম্পার কেবলমাত্র আক্রান্ত মানুষটিরও হয়না, তার পরিবারের লোকজনদেরও একধরনের মানসিক-শারীরিক-অর্থনৈতিক ক্যাম্পার গ্রাস করে। সেই সব মানুষদের নানা সমস্যা, সমস্যা থেকে উত্তরণের বিকল্প ভাবনা, তাদের সুস্থ ও সমাজের মূল স্রোতের মধ্যে আবার ফিরে আসা ইত্যাদি নানা ভাবনা চিন্তা এই Research Model -র মধ্যে আমরা ধরতে চাই।

শুধুমাত্র প্রতিশ্রুতি নয়, এগুলি আমাদের স্বপ্ন। কঠোর সমালোচনা নয়, আমাদের সাহসী পদক্ষেপ আপনার পরিবারটিকে আলো দিতে পারে - একথা মনে রাখবেন। তাই বড় মন নিয়ে আপনারা এগিয়ে আসুন, আপনাদের সাদরে বরণ করে নেব। কোনরকম স্বার্থ নিয়ে আসবেন না, স্বার্থসিদ্ধির সোপান নয় এটি। আপনার -আমার সহযোগিতায় একদিন এদের পাশে দাঁড়াতে পারার জন্য আনন্দ লাভ করতে পারবেন। ভুল কিছু হলে সদর্থক ভাবে শুধরে দেবেন। ভবিষ্যতে এটিকে অর্থাৎ 'গান্ধি সেবা সংঘ'-কে সামনে রেখেই যেন আমাদের উদ্দেশ্য সফল হয় সেই আশীর্বাদ টুকু একান্তভাবে কামনা করি।

ডাঃ অসীম চ্যাটার্জী —

About “Combat Cancer”

Incidentally during my tenure as a chief coordinator of the Subodh Mitra Cancer Hospital, I came into close contact with monk, Vitthal Maharaj, who happened to be the Principal of Thakur Omkarnath Ashram. He mentioned that a British-sponsored NGO under the slogan, COMBAT CANCER, had expressed their intention to render honorary services for the downtrodden people in the villages of West Bengal. They proposed to donate a modern well-furnished mobile Van with all equipments and arrangements necessary for early detection of cancer. The proposal was highly acclaimed and accordingly a survey was made in the villages of three districts of West Bengal viz. Howrah, North 24 Parganas and South 24 parganas regarding the incidence of cancer specially among the poor people. But for the presence of foreigners like British personnel, controversy arose regarding the modus operandi of carrying out the project. It could not be resolved unanimously and as a result the project was finally dropped. We, however, did not relent; rather tried to keep space in our routine rather work. After a few days under my convenorship a meeting was convened in the University Institute Hall at Kolkata to discuss an agenda on ‘Cancer Treatment for All’. The hall was full with dignitaries. A large number of enthusiastic participants comprised of the then Mayor of Calcutta Corporation, reputed oncologists, general physicians, research scholars, social activists and others thronged the Hall. Keen interest among them was distinctly visible. But doubts were raised about the practicability or feasibility of the slogan ‘Cancer Treatment for All’. Owing to diverse opinion of the speakers, associated with the issue of financial constraints, no concrete programme was spelt out. And for the time being this stupendous effort did not materialize. From the very beginning our objectives or aims were moulded by the broader social perspective. We pledged that all of our work should be challenging and we should concentrate on general public so that its outcome may be universal. In Subodh Mitra Cancer Hospital, from the very beginning I was conscious enough to avoid the influence of partisan politics and tried to use the ambience of the region where I lived and connected my projects with the medical centres of that region.

South Dum Dum Municipality area where I reside, is quite a diversified region. Inhabitants include businessmen, wealthy people and intellectual people on one side, while huge slum and colony areas exist on the other side. We tried to carry out a survey of this area and arrange for treatment of Breast & Cervix cancer for socially backward and economically weaker section of the people. We aimed to utilize the cooperation of Subodh Mitra Cancer Hospital to extend the facilities of government hospitals to the cancer patients of this region. Practically we wanted to establish a module for early detection and treating cancer right at South Dum Dum Municipality area, so that this could be provided later on to other Municipal areas as well. We proposed it in May 2008, and Chairman Shrehir Bhattacharya agreed to cooperate at their best. A letter of request from Municipality in this regard is enclosed herewith. Unfortunately thereafter we had to face opposition and criticism from different sections of people for this project. Ultimately the project had to be abandoned.

Dr. Rabindranath Chatterjee Memorial Cancer Trust

When we were trying to establish a centre exclusively for cancer detection, treatment and research, we first thought of registering it under the Societies Registration Act, but the Chief Medical Officer of Barasat said that it would not be possible to give permission of building such an institution. Although he knew me well and had no doubts about my abilities and honesty, my papers having been published in many international journals people knew and recognized me, so, however, he advised that I should take up the proprietorship of the institution instead of creating a society first. In his opinion, this might also affect my research work and obtaining permissions would be difficult. At the most under such a situation, as he viewed, a Cancer Centre like a day-care should better be created. He advised me to build this institution by the name, Critical Cancer Management and Research Centre and Clinic (CCMRCC) first and whenever I would feel the need of formation of a society to run and administer this institution, required permissions could well be obtained. The society would act only as a supplementary body, not as the core management body, because it would again have its own constraints. I discussed this issue with my colleagues and well-wishers and they gave their acceptance to this proposal of creating a Trust. Mr. Shankar Chakraborty was very excited regarding this new venture and seconded the proposal. He was given the responsibility of creating the Trust and was free to take any decision in this regard. A committee was formed which suggested that this Trust be named after my father. I did not quite agree to this at the beginning; however, later on I had to. Under the active guidance of Mr. Shankar Chakraborty, within a few days Dr. Rabindranath Chatterjee Memorial Cancer Trust was formed. I was appointed the Secretary and my son Dr. Aradeep Chatterjee became the Joint-Secretary. Prof. R. S. Bhakta was appointed the President and his brother Mr. G. S. Bhakta, a Chartered Accountant by profession, was appointed the Treasurer of the Trust. We requested Mr. Shankar Chakraborty to remain a part of the Trust, but he firmly declined. We applied for an exemption under Section 80G of the Income Tax Act for the Trust and got it without much difficulty.

However, after a few days we lost Prof. R. S. Bhakta, Mr., Deb, Vice-President of the Trust. To fill in those positions we are in constant persuasion with Mr. P. Chakraborty, ex-Principal Advisor (Law and Judicial) and ex-Principal Secretary, Mizoram Government, and Mr. Pushpendra Krishna Bhowmick, an economist.

Establishment Of Our Own Cancer Research Centre

An urge for setting up of a Cancer Research Centre-cum-hospital of our own had all along been in my mind for last so many years. I became almost restless. Meanwhile, the HIDCO Chairman and a few ministers of the West Bengal government decided to give us land and help us set up a Cancer Research Unit at Rajarhat. But due to a difference of opinion with my colleagues and disapproval from some of my staff against the ruling party in the West Bengal Government, I declined to accept the offer along with all kinds of financial assistance. After sometime we created a new NGO at my residence, named “New Horizon Centre for Cancer Research and Treatment”, as I have mentioned earlier.

The Cancer Institute at 381, S.K. Dev Road, Kolkata-48, had initially functioned through 'ONCOLINK' for almost 5 years. As already stated above, the new scientific forum, 'New Horizon Centre for Cancer Research and Treatment', also joined hands with them. Dr. Subir Ganguly, then President of IMA (Indian Medical Association) and Dr. Prabir Sur, the then secretary, IMA, Calcutta Chapter came forward to help me out and were very much particular in detailing out my work. But our work got severely affected as soon as they moved to other places on transfer. Luckily, Prof. Anup Majumdar (the then Head of the Oncology Dept. of SSKM Hospital) extended full cooperation to the 'New Horizon Centre for Cancer Research and Treatment' which was later renamed as Critical Cancer Management and Research Centre and Clinic (CCMRCC) as stated earlier.

Talking about our own Critical Cancer Management and Research Centre & Clinic (CCMRCC), the facility was initiated with only one bed which too was donated by Shri Shankar Chakraborty. His elder brother Shri Sudhanshu Sekhar Chakraborty was a social worker. Once both the brothers arrived, Sudhanshu-babu mockingly said that both, he and his younger brother, were unmarried and if they be victims of Cancer, they would like to get admitted at the CCMRCC. As fate could have it, just after fifteen days Shri Sudhanshu Sekhar Chakraborty was detected with Cancer and was admitted in this very facility-Centre. He eventually became our first patient.

Our second patient admitted to our facility Centre turned out to be Mrs. Munmun Ganguly's father. Mrs. Munmun Ganguly was associated with us for a long time. At that time we did not have either a Cancer unit or any license for it. For Mrs. Munmun Ganguly's father we arranged a folding camp-bed. Prof. R. S. Bhakta said that it was not only improper but also unethical, and advised us not to continue with it anymore. Discussions started between Shri. Shankar Chakraborty and Shri. Saurav Chakraborty (Chanu). They said that we all should now think big for setting up of an authorised Cancer unit. The then CMO (Chief Medical Officer) was an acquaintance of said Saurav Chakraborty. He described the project as a very risky one. We went to the Pollution Control Board to obtain permission. While I was in their office, a person came forward and took me to a room and made me seated there. I was amazed and told him the reason behind our visit to the Pollution Control Board office. He later said that he knew me and had been to my house once to visit his relative who had been under my treatment. He further conveyed that he knew that I had once served as the Chief-Coordinator at Subodh Mitra Cancer Hospital, Kolkata. He assured me of all his help to get the required permission, although it was not his department. Consequently all the related enquiries were held and within fifteen days we got the State Pollution Control Board's permission of installing six beds. Then I went for and obtained the hospital license. Gradually we built an OT (Operation Theatre). Later, to manage huge pressure from patients seeking admission, in the first floor four more beds got the sanction.

From the very beginning, as already mentioned, I had started working with mainly those patients who were very very poor and terminally ill, and of course with their express consent. Since I started with socio-economically backward people, so in the initial or primary phase I used to get not many patients. The reason behind this being that I lacked in experience. I was believed to have very minimal knowledge of detecting cancer accurately as to whether

a patient is suffering from the disease or not. Secondly, at that point of time technique of primary detection of cancer was not so developed as it is today. Because of this I had to work with patients who were at an advanced stage of the disease. In most of the cases, I had to bear the total expense of their treatment since they lacked in any financial support, and at the same time they were very less in number. But today I treat a large number of patients and my cancer related knowledge has also become rich. I gathered huge practical knowledge while treating a large number of patients both at my house and at my institute. As a result, now we are able to provide much better service to those who come from economically poor background. But the main problem that stands as a barrier is finance the everyday-changing laws and legal system, and the complexities related to it. However, we have addressed the problem of our financial constraint in a different way. Our hospital, CCMRCC, acts as a source of revenue which takes care of our financial commitment towards the poor to a large extent. We also started initiatives getting permission to give subsidized treatment to the poor. We also resolved documentation and preservation of all our experimental works, experience, achievements and recognition so that it might help the next generations to view a new horizon of cancer-cure.

We started our hospital with a whole new perspective, with an objective to innovate a new model that would be acceptable to the entire society and something that would benefit the entire mankind. I started the service with less experienced people employed at my hospital. They were not trained in any big hospital or Nursing Home. I got them trained, because I wanted to treat the patients by rendering all the services and care to the patients 24X7 hours, and by involving everybody in the family and around. It will in due course educate everyone associated with me including the patients' family-members. For initial few days we got satisfactory results, but soon I found that we were improvising personnel management in our own typical way in a highly technical and sensitive area i.e. health care. Anyway, due to this, two new problems cropped up. Firstly, the efficiency that we expect from trained people went on missing. Secondly, the unskilled people have many unjust expectations and less social commitment. Sadly enough, I had to let a few of my employees quit, but the people who still remained with us, their social commitment stood the test of time and was exemplary. Consequently, at a later stage those people grew much more experienced and proved their expertise day-by-day.

Now that everything was going as expected, we started thinking of a new model and to work towards its formal shaping. I had too many expectations from this second venture. We sat in discussion with experienced personalities whom I was privileged enough to get associated with at some point of time in my 35-year-long journey, in order to create a future road-map. These people were known experts in their own fields, who had always supported me and were my advisers and friends. In the discussion, Prof. Hiranmay Mukherjee came up with a relevant point that it would be a challenging task to create a second model effectively and efficiently, and further said: ***It must not be any of the Homeopathy, Ayurveda or Unani centres. If it were anything of that, we would not have come. Here we all have gathered to discuss a major problem concerning people from all spheres. We have to gain confidence of the common people and the medical fraternity as well in order to proceed further and to create a new horizon.*** Another question came up as to who will take the

lead and become the key person in respect of the second model. At first we were supposed to appoint competent person who would be capable enough to bear the responsibility and also find answers to questions that why would the experts consult him and why would the common people keep faith in him. Dr. Mukherjee added that when the journalists would ask them about the efficacy of the treatment in this new model, they would not be able to justify. Although my existing work had been certified, Prof. Anup Majumdar said that the results had been encouraging in respect of the first model but its technology was hardly known. He further said: *This new model is going to have I.C.C.U., operation theatre, facilities like radiotherapy and chemotherapy, doctors of medicine etc. but what is more important is the presence of Dr. Asim Chatterjee. He has a far more clear perception that he has gained over the years, compared to anybody else. When he invites Oncologists to administer Chemotherapy, they respond without any hesitation. Onco-surgeons come and perform surgery and also take opinion of Dr. Chatterjee. Surgeons usually think that radical surgery is more effective. However, on Dr. Chatterjee's suggestion palliative surgery is given priority over radical surgery. The reason being, majority of patients treated here come from a very poor background, live in villages, and in most cases are aged and are at the terminal stage. They opt for palliative surgery because they feel that it would help them better. But none of us is quite aware of the finesse of the technology involved in palliative surgery. Although Dr. Chatterjee is not an onco-surgeon, still he has a clear concept related to surgery and what type of treatment to be administered on to which patient. The onco-surgeons and chemotherapists also have full faith in him. Now if the Psorinum therapy cannot be dovetailed with the ongoing treatment methodology, the treatment will not leave any overall beneficial impact and this is not going to be acceptable to anyone. The prospect of creating a second model of Psorinum therapy would compel us to open the nucleus of the technology of Psorinum therapy and the management associated with it, and how it would become a part of the 'Chikitsa Shastra' etc. would necessitate absolute clarity. To expect this kind of clarity of concept, vision and commitment from somebody else would be futile. So the second model would have to remain in our imagination for some time now.* Dr. Mukherjee also stated: *I have guided many Ph.D. students and I know the research related methodology. So here the important question is what is Psorinum therapy? In order to create a second model this question has to be answered first. The work has been certified, not the method.*

Be that as it might have been, my innate happiness derived from my research-drug, the therapy and the extent of care of the patients by all of us including my family-members nevertheless did recede ever. Perhaps, the Critical Cancer Management and Research Centre & Clinic (CCMRCC) is the only cancer-patients' care-centre which is under the same roof of the physician's whole-time residence and provides the very personal care and treatment round the clock. It is my wife, Ranjana, who with all-the-time smiling face is in the sole charge of the hospital administration. Our services to the patients' over-all treatment include honest and selfless counselling to their family-members too. When we treat patients, we also look into their family problems and consider their financial issues, the patients' own needs and requirements. We have seen people dying with money in their bank accounts or lent to others, of which their family members sometimes had no knowledge and a result had to lose on many occasions. We try to inform all the patients about that and make them aware. Generally

patients suffering from cancer related to lung, liver, gall bladder, stomach, pancreas and GB do not live for long. Patients who are relatively young are advised to undergo counselling sessions. We try to make them understand the crisis and to wisely take each and every step and to keep them prepared from all angles including family-settlement and property transfers etc. Especially business people are asked if they have given any loans to anybody, they should take initiatives to recover it from the borrowers, and the like. This is done because we have come across cases where families of a few businessmen had to suffer huge losses and to face innumerable problems after the patient's death, in no time. In the next volume we will talk about these aspects in more details.

In many cases, we see that some patients whose children are in their marriage-age, wish to see them getting married, settled and happy in their family life. In those situations to save or to stretch their lives even for a few more days becomes a real challenge to us. We have come across almost 50 such cases where we have tried to make the patient live for as many more days as possible so that he/she can see their children getting married. Surprising but true, in at least 40 such cases we have succeeded. Similarly, in six months we arranged six marriages. I am providing here a few such cases with detailed description for the readers to understand the situation better.

As I have stated, marriage plays a very important role in a cancer patient's own as well as family life, which also affects his/her psychology. Cancer implies that one very important component of the family is soon parting away from the rest and in this situation the cancer-sufferer wants to see everything well and good in his family before breathing his/her last. The lady patient who is a housewife, wishes to see her place being filled by the new-comer in the family, i.e. her daughter-in-law. Again, a man suffering from the disease, who is the head of the family, wants her daughter get married and live a happy married life and his prospective son-in-law to be there to look after his family, if needed after he expires, just like a son. The new addition to the family also helps others to overcome the grief of losing someone. This is how a marriage can change the tragic scenario in a cancer patient's life and of the affected household to some extent. Similarly we need to keep so many things in mind while treating a cancer patient; we try to relate to them and become a part of their family and make them share their anxiety.


I would like to present here three such cases, as follows:

Firstly, there came a patient named Smt. Rikta Paul who was only 50 year old. She had cancer in her thyroid gland and it was in an advanced stage. The cancer had also spread to her lungs, liver and bones. Her only son, a meritorious student and an M. Tech. from IIT (Kharagpur), is in the academic profession. Everyone in their house had lost all hopes of the patient's survival. During their counselling I told them that death is inevitable, and suggested that it would be better if there be a new addition to their family in the form of their daughter-in-law. At the beginning they were not in agreement with me but later as the patient's condition started failing, the patient herself expressed her desire that before dying she would like to see her son get married, and to give away all her responsibilities to her daughter-in-law. Then only she would feel happy and relieved. The family started looking for a suitable girl and within two months the wedding took place. The patient then spent 15 days

with her family at her residence. After that she was re-admitted at my facility, CCMRCC, where she passed away within next few days. The family is still in our close contact and invites us during any ceremony or event performed in their house.

Reports and supporting documents of Smt. Rikta Paul.

Discharge Summary :: http://192.168.1.100/HMS_Thakurpukur/modules/help_desk_un/print



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 E-mail : cancercentre6@gmail.com / cancerwelfare@yahoo.co.in Website : www.cancercentrecalcutta.org

DISCHARGE SUMMARY

Name	:RIKTA PAUL	UHID	:201501044G
Age/Sex	:62Y 8D/Female	Admission No.	:2015001000
Father/Spouse Name	:LATE BANKIM BEHARI DAS	Dept.	:RADIOTHERAPY
Visiting Doctor	:DR. MAHFUZ ARIFF	Bed No:	:15
Ward	:LINAC		
Tel	:8017275423		

Date Of Admission	: 05/02/2015	13:17 Admitting Doctor	: Dr. MAHFUZ ARIFF
Date of Discharge	: 13/02/2015		

Summary
 A case of multiple SOL in lung, liver brain, liver , brain, multiple ↑FDG uptake area in bone, brain. She had follicular neoplasm 2005. Now she had fracture humer (Rt). Trail of IM nailing outside has failed. Board decided for trucut biopsy of lung/mediastinal SOL.

Final Diagnosis
 Metastatic carcinoma primary thyroid or lung.

Investigation
 All report enclosed.

Advice On Discharge :

2/13/2015 1:13 PM

The second case to talk about is Smt. Jharna Saha from Kamarpara, Jalpaiguri in West Bengal. She was a 48 year old lady who was brought to us in a very serious condition. Her platelet count was 3000, haemoglobin level 4.6 and WBC count was 2000. She was a post-treatment patient (Tata Medical Centre, Rajarhat) and was admitted to my facility on 22.01.2015. The only request she made was that she wished to witness her only daughter's marriage on 27.02.2015. The patient-party paid for the hospital and medicinal expenses and returned home. They regularly kept contact with us and the patient's brother who used to

stay in Kolkata, used to visit her in my facility and keep track of overall development of her health. Fortunately enough, she could witness the wedding and on 02.03.2015 she took her last breathe.

Reports and supporting documents of Smt. Jharna Saha.



TATA MEDICAL CENTER

Tata Medical Center
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Phone: +91 33 8605 7000, 7222 , Email : info@tmckolkata.com
Website: www.tmckolkata.com

Run Date : 22/01/2015 10:47:44

Discharge Summary

MR No. : MR/14/010428
Patient No. : IP/15/000277
Address : KOLKATA 4 20, MOHANLAL STREET

Name : Mrs. JHARNA SAHA
Sex : Female
Age : 48 Y 3 M 28 D
KOLKATA, KOLKATA, WEST BENGAL-700004, INDIA

Admitting Department : Medical Oncology
Admission Date : 14/01/2015 18:36:28

Admitting Doctor : Deepak Dabkara
Discharge Date : 22/01/2015 10:30:57

DIAGNOSIS

Carcinoma of unknown primary.
On best supportive care.

Admitted with grade 4 fatigue, anemia, fever.

Discharged on request.

HISTORY

CUPS.

Slide and block review at Roy and trivedi- Metastatic PD Adenocarcinoma.

Post 2 cycles of chemotherapy and palliative RT (Last Chemotherapy with single agent peditaxel on 17.11.14).

Had recurrent Myelosuppression during last chemotherapy cycles.

In view of poor tolerance to chemotherapy (Had recurrent pancytopenia after chemotherapy) cancer chemotherapy stopped and planned for best supportive care.

Admitted with severe weakness, anaemia, thrombocytopenia, fever.

ON EXAMINATION

On admission-
PS 4
Pallor ++

INVESTIGATION RESULTS

CBC+DIFF (19/01/2015 04:35:06)

WBC : 3800 , HGB : 5.5 , PLT : 6000 , MPV : 8.3 , NE% : 86 , NE# : 3268 , LY% : 8 , LY# : 304 , MO% : 6 , EO% : 0 , BA% : 0 , RBC : 1.80 , PCV : 16.1 , MCV : 89.7 , MCH : 30.4 , MCHC : 33.9 , RDW : 19.3

ELEC/RENAL PANEL (19/01/2015 04:35:06)

UREA : 21 , CREATININE : 0.3 , SODIUM : 129 , POTASSIUM : 3.1 , CHLORIDE : 95

POTASSIUM (18/01/2015 05:01:27)

POTASSIUM : 3.4

PT WITH INR (17/01/2015 10:18:15)

TEST : 15.2 , CONTROL : 11.1 , INR : 1.36

D-DIMER (17/01/2015 10:18:15)

D-DIMER : 19782.43

APTT (17/01/2015 10:18:15)


TEST : 30.9 , CONTROL : 29.0


Page 1 of 2

Lastly, I would mention about a patient named Smt. Sarama Mondol, a 39 year old

widow suffering from colon cancer. Her family's financial condition was very lean. She lost her husband when their only daughter was hardly 2 years old. Now the girl is 18 years old and a student of Political Science in Basirhat College. She got engaged to a person from a very good family background, and a high-school teacher. Their wedding date was already finalised. When the patient was admitted here, her haemoglobin level was 4.8 and she was bleeding profusely from the rectum. Observing her condition we took initiative to get her admitted in a government hospital, but things did not turn out well. Later she was admitted to my facility on 13.1.2014. She told me that she knew, she would not survive long. She pleaded tearfully that her daughter's wedding had been scheduled sometime in next two months and she simply wished to see her daughter married. I also came to know that the lady was a resident of the same village I hailed from. We gave her eight units of blood. We also performed colostomy on the patient and sent her back home in a better condition. Then the wedding took place very nicely and we advised her to get admitted again soon after the wedding. Unfortunately, for quite some time the patient's family stopped contacting us. Nevertheless, we are happy that her wishes came true and her daughter is now leading a happy married life. In my book 'Total Strategy Against Cancer,' I have shown such case-histories dated back to 1990s. Each and every patient carries his own individual problems. We at CCMRCC, before initiating the treatment, try to unearth those issues and look for their mental resolve during the treatment.

Reports and supporting documents of Smt. Sarama Mondol.





DIAGNOSTIC CENTRE

R B Diagnostic Pvt. Ltd.

Lake View Branch
77/1, West X, Lake View
Bhubaneswar - 751005
T: 2551 888 / 467 761, R: 1838 4331
rbdiagnostic@gmail.com, www.rbdiagnostic.com

Sat Lake Branch
G-45, Sat Lake
Bhubaneswar - 751004

PA1 No. : 1027

MONDAL

AGE/SEX : 44, male

REF. DOC : DR ASIM CHATTERJEE

RECEIVED : 18.12.2014 10:20 AM

REPORTED : 18.12.2014 04:27 PM

CENTER : A-OPD

Page : III

Urinary bladder :

Bladder is normal in contour without any focal abnormalities in its wall. There is no papillary growth nor any intravesical abnormalities. Perivisceral fat planes are normal.

Uterus & Adnexa :

Uterus appears normal in shape, and size for this age. There is no focal detectable mass in uterus nor in adnexa.

IMPRESSION : CT scan of whole abdomen revealed :

Rectal and rectosigmoid large ulceroproliferative stenosing growth with perirectal lymphadenopathy - P CA Rectum.


Right renal benign cortical and simple cyst.

Please correlate clinically.


Machine Details:

Spiral CT scan; 16 slice Bravo 385

Low radiation dosage technology.

Checked by 

End of Report



DR S L KEDIA
MD (Radiodiagnosis), PGD
(Chandigarh)

This laboratory finding is only intended as a supplement to clinical presentation & related investigations as referred purposes. The reported results are dependent on individual case procedures, method, specificity & sensitivity, equipment used and quality of specimen received, and operator. This investigation report is not valid for medico-legal purposes. For any kind of technical queries related to the report, please contact us immediately.

For pathology sample collection & report delivery at your doorstep, Call 12221 0038

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e-mail : unimedkolkata@gmail.com



REF. NO : L-4084 (OS)

DATE : 14-12-2014

NAME : Ms. Sarama Mondal (OS)

SEX : F AGE : 39 YRS

Referred By : Dr. Ashim Chatterjee

H A E M A T O L O G Y

HAEMOGLOBIN : 4.8 gm/dL (33.1%) PCV : BLEEDING TIME (Ivy's method) :
(Cyanmethaemoglobin) [Male : 13.5 - 18 gm/dL]
[Female : 11.5 - 16.4 gm/dL]

MCV : [2-7 mins]

RBC : MCH : CLOTTING TIME (Lee and White):

PLATELET : 4.75 lakhs/c.m.m. MCHC : [4-9 mins]

RETICULOCYTE COUNT : RDW :

DIFFERENTIAL COUNT: Abs. Count Abs. Count

Neutrophil	: 64 %	5824 /c.m.m.	Blasts	: 0 %
Lymphocyte	: 30 %	2730 /c.m.m.	Promyelocyte	: 0 %
Monocyte	: 1 %	91 /c.m.m.	Myelocyte	: 0 %
Eosinophil	: 5 %	455 /c.m.m.	Metamyelocyte	: 0 %
Basophil	: 0 %		Band Form	: 0 %

E.S.R.

1st Hour :

FILM MORPHOLOGY

RBC : Hypochromia(++), Anisocytosis(++), Poikilocytosis(+)
Tear droplets(+), Target cell - Occasional

WBC :

Platelets :

Parasites :

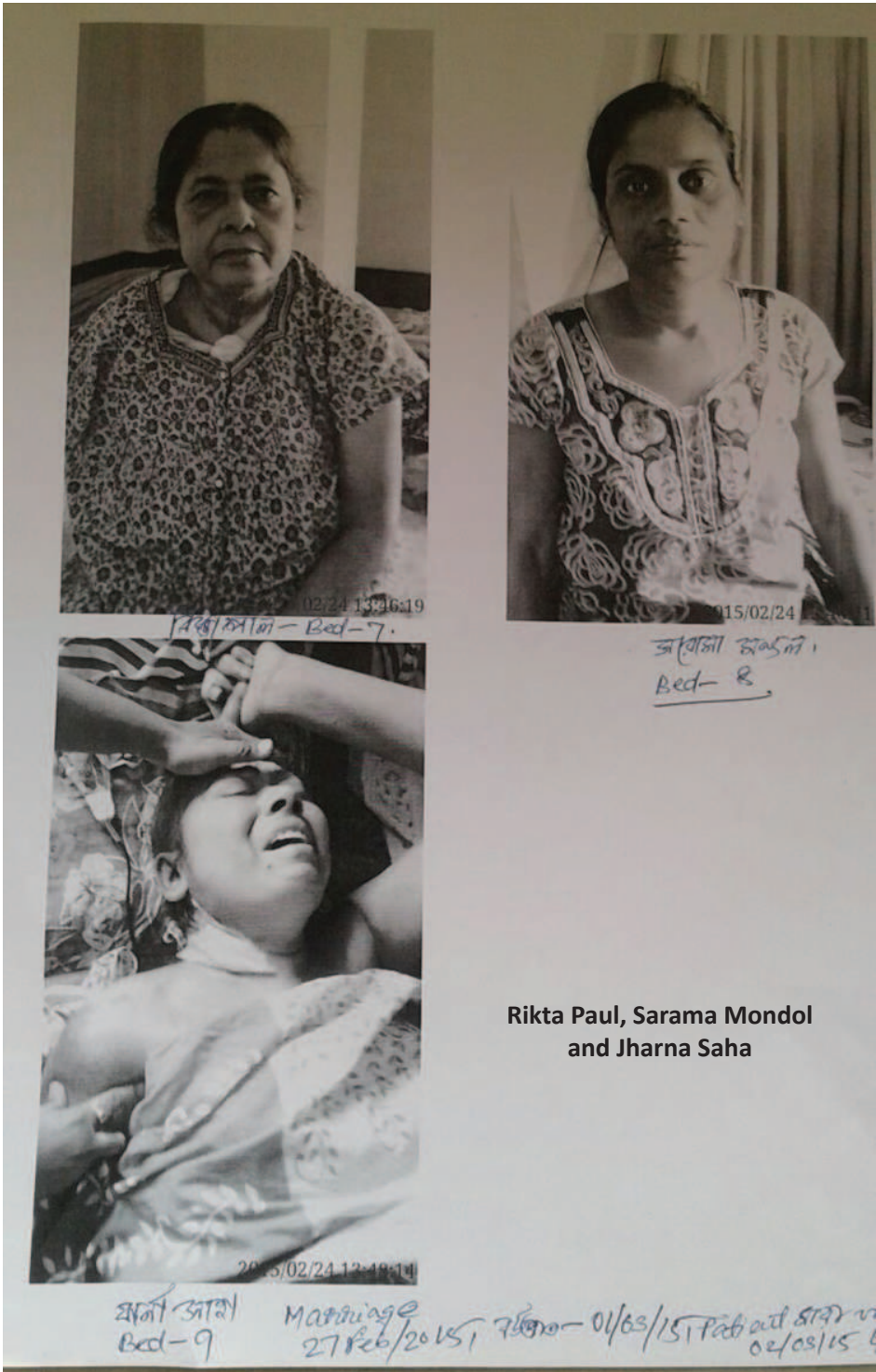
Checked By AS

Dr. A. Mukherjee
MD (Path)
Consultant Pathologist

Dr. S. Gupta
DCP, MD (Cell)
Consultant Pathologist

Dr. Goutam Bandyopadhyay
MD(Path) (AIIMS-New Delhi)
Prof. R.G.Kar MCH.

Mr. S.R. Sarkar
MSC (Biochemistry)
Biochemist



Rikta Paul, Sarama Mondol
and Jharna Saha



Prof. Anup Majumdar visiting a patient at CCMRCC



Prof. S. Saha in the OT of CCMRCC

A few days ago a patient was brought to our Facility. She belonged to an influential family of Kolkata. She was accompanied by her husband, a Chartered Accountant. The lady had colon cancer and also suffered from multiple metastasis of liver. She had already gone through colostomy i.e., superficially taking out of stool from her body without undergoing a colon surgery, from a renowned hospital of Kolkata. Lately they were advised that the patient would require three chemotherapy sessions to be followed by a major surgery on colon. These times are very crucial, emotional and indecisive, both for the patients as well as for their near and dear ones. The patient's husband asked me only one question at that point of time: Will

my wife survive for next 10 years? I could easily relate what the person was going through at that point of time. I suggested to him to undergo counselling, two hours daily, for next four days. He attended thorough counselling sessions to understand many things related to the subject and was told that it would be really difficult for the patient to survive even for one year. On the fourth day the patient's husband admitted that he had understood a few things. First, at any cost nobody would be able to make his wife live for more than a year. Secondly, he will have to plan how he would be going to live without her, how lonely he would be, etc. Lastly, it would not be wise to spend too much on his wife's treatment as they had a daughter as well, for whose education and marriage, he would have to keep adequate fund.

Talking about the patient, liver surgery appeared to be not feasible, since it is metastasis, not primary. It is multiple metastasis; removal of one SOL (Space-Occupying Lesion) would not help and surgery was also not advisable. I asked the patient's husband to decide whether he would like to go for chemotherapy or opt for Psorinum therapy. He asked for ten days' time to think and decide. He had allocated 25 lakh rupees towards his wife's medical cost earlier, but of late he realised that it would be better that if the expenditure be minimised to 3-5 lakh rupees. I assured the patient of all the help and support possible from my side. But the patient's husband reacted to this, and said that whatever I had suggested seemed very rude and unkind to him and that he would consult other Oncologists and approach better medical institutions but won't seek my assistance any more.

Another patient was admitted to my facility some time ago. The lady is a widow and has a son who is a primary school teacher. The lady had cancer in her rectum and left lung metastasis. I told them that the rectum cannot be surgically removed and we need to perform a colostomy on the patient. Later either chemotherapy or Psorinum therapy could be applied on the patient. The patient's son said that they had complete faith in me and gave me all the liberty to do whatever I would think the best for the patient. I, by that time, was completely aware of what the patient's financial capabilities were. The patient was admitted, primary tests were performed, and after colostomy we administered the Psorinum therapy and in due course sent her back home.

Similarly, a 90 year old widow named Sudharani Dasgupta was admitted to our Facility. She was suffering from CA (Carcinoma) of lung, effusion and ascites. Earlier she had been admitted in the Calcutta Heart Clinic and Hospital, Salt Lake, where she was undergoing chest drain for some time. Daily 1.5 litres of effusion was taken out from her lungs and the colour of the fluid was red. The oncologists advised that if the quantity of effusion be brought down to 50 ml then only Pleuradesis could be performed on the patient i.e., injecting a drug in the pleura followed by joining of the lung space, to stop the formation of the fluid. The patient's condition was such that chemotherapy could not be performed on the patient. In that nursing home a physician referred the family to me and told them that her treatment would be possible under Psorinum therapy. The patient's family members requested me to reduce the quantity of fluid formation so that they can go ahead with the advised medical procedure. I said that I would try to reduce the fluid formation but it would be difficult to take the patient back home. The family members objected vehemently, to which I said: ***The patient may not die due to cancer only, but due to various infections, age and other associated***

causes. The family members realised that that I was right and the patient was admitted in the recovery unit of CCMRCC. Very soon the effusion completely stopped, but since the patient had diabetes, she suffered from multiple infections one after the other. One of her lungs also collapsed. It was by then 45 days since she had been admitted at the CCMRCC. Unfortunately the patient developed nutritional loss due to lack of appetite. Owing to all these factors, the patient's condition got deteriorated. Although the effusion had stopped, she could not be allowed to go back home. There was an expenditure of 2 lakh rupees till then. I told the patient party that to make the patient live for two more months, it would involve an expenditure of 3 lakh rupees. The patient party agreed and said that they wanted to save her whatever might be the cost. To control the nutritional loss the patient was kept on a substitute diet like fat soluble, human albumin, etc., and also on antibiotics which were very costly. But as luck could have it, the patient died of a massive cardiac arrest.

Reports and supporting documents of Sudharani Dasgupta.

CALCUTTA HEART CLINIC & HOSPITAL

A Non Commercial Medical Centre under W. B. Societies Registration Act. 1961
HC BLOCK SECTOR III BIDHANNAGAR KOLKATA 700 106 INDIA
PHONE : 23585735 / 23587885 / 23214578 / 2334 5144 ; FAX : (033) 2358 8876
E-MAIL : calheart@vsnl.net Website : www.calcuttaheartclinic.org
REGISTRATION NO. S/16611 OF 1975-76

Name : Sudha Rani Dasgupta Age : 90 Years Sex : F
Address : Indoor, Bed No. CCU-104 Dated : 31.07.15
Ref. By : Dr. N.R. Naiya

ULTRASONOGRAPHY OF UPPER ABDOMEN& CHEST

(Portable)

Liver : Mildly enlarged in size. (size 136 mm.); coarse echotexture.
Intrahepatic biliary ducts are not dilated.
No focal S.O.L. noted.

Gall Bladder : Wall not thickened. Lumen appears clear.
Pericholecystic area clear.

Common Bile Duct : Not dilated. Normal in size. (Size 5 mm.)

Portal Vein : Not dilated. Normal in size. (Size 8 mm.)

Pancreas : Normal in size; parenchymal echogenicity increased,
suggestive of fatty change.

Spleen : Normal in size. Normal in size. ((Size 66 mm.)
Parenchyma appears homogenous.

Both Kidneys : Right kidney 83 mm. Left kidney 89 mm.
Shape, position normal.
Corticomedullary differentiation normal.
No calculus, no S.O.L., no hydronephrotic changes seen.

* No ascites noted.

Hemithorax :

Large, anechoic, pleural effusion noted on the right side extending
superiorly to the upper margin & pleura.
Collapsed lung tissue seen within the effusion.
Left costophrenic angle clear.

IMPRESSION :
1. Mildly enlarged, coarse liver.
2. Fatty change pancreas.
3. Large, anechoic right pleural effusion


Dr. S. Mukherjee
MBBS, DMRD
Consultant Radiologist
Calcutta Heart Clinic & Hospital

AMRI-DEPARTMENT OF LABORATORY MEDICINE

NABL ACCREDITED (ISO 15189:2007) Certificate No. M-0162

CYTOLOGY

Patient Name : Mrs. Sudha Rani Dasgupta
Patient ID : FR00049874
Location : Diagnostic Clinic
Doctor : Referral Doctor
Order No : LBOP00000190435
Specimen No : 5515000873
Payer :

Age/ Sex : 90Y /FEMALE
Bed Number :
Ordered : 31/07/2015 21:51
Collected : 31/07/2015 22:51
Received : 31/07/2015 22:51
Reported : 03/08/2015 11:03



Certificate No. M-01

Specimen Type : SMEAR
PAP SMEAR

PLEURAL FLUID FOR CYTOLOGICAL EXAMINATION


Microscopy :

Smears show malignant cells in clusters with acinar pattern and dispersed singly. Cells have hyperchromatic nuclei & moderate amount of cytoplasm. Background shows lymphocytes, macrophages & reactive mesothelial cells.

Impression : Metastatic adenocarcinoma

Slides : 02
FC No. : 883/ 15

--- End of Report ---


Dr. Hema Chakraborty
DCD, MD
Consultant Histo & Cytopathology

Dr. Anil Kumar Saha
MBBCh (UK)
Consultant Histo & Cytopathology

Dr. Shrivastava
MBBCh (UK)
Consultant Histo & Cytopathology

Dr. Sangham Patra M.D
(Consultant Pathologist)

Dr. Sanghamitra Mukherjee M.D
(Consultant Pathologist)

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E-MAIL : calheart@vsnl.net Website : www.calcuttaheartclinic.org
REGISTRATION NO. S/16611 OF 1975-76

Name: *Sudha Rani Dasgupta* Age : *90 years* Sex : *F*
Address : *Indoor, ICU-7*
Referred *Dr.Saibal Ghosh* Date: *03.08.15*

H.R.C.T. OF THORAX

PROCEDURE:

Plain high resolution C.T. scan of thorax done by taking 2 mm. thin slices at 7 mm. intervals from lung apex to domes of diaphragm.

DIGITAL RADIOGRAPH:

Digital radiograph of supine AP projection of chest shows central mediastinum with cardiomegaly. Normal domes of diaphragm. Lung fields expanded with patchy opacities in right side.

CHEST WALL & BONES:

Bones under review show intact cortical outline. Paravertebral & parietal muscles are normal. Axillary lymph nodes are not enlarged. Osteopaenia in bones with degenerative changes in spine.

MEDIASTINUM:

Mediastinum is central. Trachea, right and left main bronchi are patent. Carina is normal. Great vessels of Mediastinum are normal. Pulmonary artery trunk is not dilated. Cardiac silhouette is normal with no pericardial effusion. Small mediastinal nodes seen in pretracheal, right tracheobronchial region. Hilar clear with no SOL both sides.

LUNGS:

Lung fields both side expanded with pneumonitis and ground glass haziness all segments right upper lobe, lateral segment of right middle lobe.

Consolidation in right lower lobe apical segment and posterior basal segment of both lower lobes. No sizeable SOL or bronchiectasis seen.


PLEURA:

Pneumothorax appearing encysted seen along right anterior and lateral costal wall in upper and mid zone. Mild pleural thickening seen along both posterior costal walls in right mid and both lower zone. Pleural minimal effusion seen right side lower zone and extending into oblique fissure.

IMPRESSION :- H.R.C.T. scan of thorax reveal:

- 1) *Pneumothorax appearing encysted seen along right anterior and lateral costal wall in upper and mid zone.*
- 2) *Consolidation in right lower lobe apical segment and posterior basal segment of both lower lobes.*
- 3) *Mild pleural thickening both sides with effusion seen in right lower zone.*
- 4) *Non specific pneumonitis with ground glass haziness all segments of right upper lobe and lateral segments of right middle lobe.*

sc


Dr. Gautam Ghosh, MD.
Consultant Radiologist

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 REGISTRATION NO. S/16611 OF 1975-76

DISCHARGE AGAINST MEDICAL ADVICE

Unit CCT (NRN) INP No. 01-15-010322
 Name Sudharani Dasgupta Age 90 yrs Sex F Bed No. ICU-7
 Address Flat - 224, BD-45, Neelkamal Apartment, Kestopur,
Kolkata - 700101
 Date of Admission 31.7.15 at 10:26 AM
 Date of DAMA 6.8.15 at 1:00 PM

• Provisional Diagnosis

Right Sided massive pleural effusion & metastatic
Adeno carcinoma, Hypothyroid, LRTI

• Treatment received

1. Inj ZOSTUM (3 gm) bid TDS
2. Tab. ELTROXIN (100 mcg) 1 tab. ODAC
3. Tab. ERLOTINIB (150 mg) 1 tab. OD.
4. Tab. PANTODAC (40 mg) 1 tab. ODAC.
5. Syp. DUPHALAC 15 ml. at H.S
6. Tab. DOMSTAL (50 mg) TIDAC
7. Inj CLEXANE (40 mg) OD, s/c.
8. Pt discharge with chest drain, Foley Catheter
 and peripheral i.v. canula. at the time of discharge.

Medical Officer
 Calcutta Heart Clin
 and Hospital

Date 6.8.15

P. Sarkar 6/8
 Signature of RMO

Patient is leaving this hospital at his own risk against medical advice.

Date & Time 6.8.15 1 PM

Signature of Patient

from daughter
 Patient's Relative (Relational) Da

The question here is, considering the current economic and demographic scenario in a country like India, is it really feasible to spend lakhs and lakhs of rupees in such cases? Is this kind of spending justified?

The patients treated till now at the Critical Cancer Management and Research Centre & Clinic (CCMRCC) are around 6000 in number, of which around 2500 were admitted to this facility for regular observation and care. Each and every patient's medical file has been preserved here containing the notes/ records of the type of treatment they were given, their socio-economic status, counselling of their families, how we helped the expired patient's family towards rehabilitation, and overcome the situation, visible improvement of their psychological condition, and above all how the patients and their families seemed truly benefitted. The CCMRCC has been providing perhaps the cheapest cancer-treatment endowed with homely care to the cancer patients mostly of terminal stage and economically vulnerable.

Changing Scenario Of Cancer Treatment

We may have learnt a lot about cancer in the past years — but mostly what we have learnt is that cancer is a lot more complicated subject than we ever thought. It appears that we have grossly underestimated the cleverness of cancer. Cancer is the common name for about 200 diseases in which cells in a part of our body begin to grow beyond control. Although there are many kinds of cancer, still they all start because abnormal cells grow beyond control. Cancer is not just one disease — it's an octopus with many diseases gathered under a single umbrella. There are multiple levels of complexity. We have different basic types, like colon cancer versus lymphoma. Then we have hundreds of different types of lymphomas, and then every single person's lymphoma is again different at the molecular level. Even though one thinks that cancer cells are all identical, the reality is that “not every cancer is same; there are many differences.” When you attack a tumour, “you're killing a diverse population” of cells, some of which will be resistant. Most of the cancers under treatment have 100 different mutations within a single tumour and it is very difficult to model that. Moreover, we have to kill the tumour without killing the person who has it. Again, cancer cells are bi-directional, meaning that stem cells can be differentiated in mature tumour cells, while mature tumour cells can also turn back into stem cells. Thus, treatments that have involved just killing of stem cells with the hope that this would keep the cancer away from recurrence, have failed, because the tumour can always repopulate with more stem cells. Again cancer cannot be diagnosed very accurately only by a single test. Multiple laboratory and imaging tests are needed to detect cancer finally, and all these tests are not only costly but also detrimental sometimes to the existing health condition of the patient.

There are plenty of drugs that do a great job of shrinking tumours in mice, but finally turn out not to be suitable for humans. Problem is that about 95 percent of those new drug applications generally fail. 70 percent of them fail even in Stage-One Cancer cases; then remain only 25 percent. And then 60 percent of those ultimately fail, and that too, most of the time, because the drugs don't work. Some of the drugs are toxic and severely affect the patient, and it's more tragic when the drug doesn't work even after its application over a good number of days.

The treatment for CANCER can be usually made by the following methods:

1. Chemotherapy;
2. Radiotherapy;
3. Surgery + chemotherapy;
4. Chemo + radiotherapy; and
5. In worst cases - by stem cell transplant.

Unfortunately, cancer being a silent killer is detected at a much later stage, after the vital organs of a patient have already been affected, so that some time the possibility of surgery is overruled. In many cases, cancer cells become resistant to either chemotherapy or radiotherapy or both. Again, at an advanced stage or for physically weak patients radiotherapy is not advised even though the cancer type is sensitive to chemotherapy or radiotherapy. As much we have progressed technically in the treatment of cancer, at the same time the entire process has become so much expensive that the same is unaffordable by many.

The treatment protocol followed in our country has been derived from the developed western countries and suits our conditions to a very lesser extent, and a handful of people can derive utility from it. If we go deep into the critical analysis, we would see that for the treatment of cancer in India, a huge amount of money is being spent out, of which a major portion is misdirected. Some are underutilised and the entire process may seem to be nothing but mockery to many.

Approximately ten years ago, I had published a paper titled 'The Management of Cancer in Totality – India can take a lead' in which I had indicated the areas that require our immediate attention. In the true sense we have to look at the problem on a panoramic view, bring it to a point and then strike it - disease, treatment, terminality and rehabilitation. We also need to keep in mind that as per the experts the disease would increase multiple times by 2025. Almost every household is likely to be affected. A country is generally a combination of millions of households, and if such a possibility comes true, then it means that the entire country would be affected. So in order to avoid that unforeseen, maybe the inevitable, we have to move away from the ongoing conventional attitude and start afresh and think of alternative treatment methods that would be entirely indigenous and involve Indian physicians and scientists.

At present the cancer related research is primarily done by the corporate sector. There has been a shift in the mindset of drug companies which used to prefer cancer drugs that could be of prolonged use by patients, so that sale of drugs case increase by quantity. Now, companies have learnt that drugs meant for one relatively tiny population of cancer patients can still make plenty of money.

That the cancer related treatment protocol would give 100% result is yet to be developed. On the other hand, for other diseases there has been a specified treatment protocol that is being followed globally and has given us fruitful results. As I mentioned at the very beginning, cancer does not differentiate between the rich and the poor or between the literate and the illiterate.

The households that are affected by Cancer suffer not only economically, but also, to a

fatal extent, psychologically. During this time friends, relatives, neighbours and social workers need to come forward to extend a helping hand and support to them quite passionately. In many cases the patient tries to hide his condition even from his own family-members. This results in delay in treatment and may not get the right advice at right time. This may lead to further aggravation of the prevailing situation on the patient's part, as worse on his family's part.

Many patients and their families often get cheated either by the Service-providers or in some cases by some fake experts or well-wishers who take advantage of the crisis in the family. During this time the patient's family members would do anything and everything for the life and well-being of the patient. Here, I would like to narrate an incident that I came across. A few years ago, I came in contact with a family. The patient's name was Smt. Shovarani Indu and she was suffering from multiple myotoma. When the patient was brought to me, she was in a good state of health. She had three sons. Her elder son was a businessman, and the other two sons were police officers. They were the residents of Shri Durga Colony, behind Dum Dum Central Jail, Kolkata. Since in multiple myotoma, chemotherapy is very effective, I advised her to go for it, along with my research-drug. I also recommended for an oncologist, namely Prof. Subir Ganguly who was then professor in Radiotherapy Department of the Medical College Hospital, Kolkata. After that, they were neither in contact with me or Prof. Subir Ganguly, nor did they continue my therapy. I doubted what could have happened to them. So one day I paid a visit to their house ignoring all the protocol. I knew, it was not proper on my part, but still I took the risk. When I reached there, I was very warmly welcome. Everything seemed fine and after some time I left. After a month or so, one relative of the patient came to meet me. He asked for my help to save the lady. Later on, what he told me was something I had never imagined. They had been seeing a 'tantrik' who was actually a fraud and promising them to cure the patient after performing some religious practices at a cremation ground. The family spent thousands of rupees in buying 2000 lotuses, 40kg. of pure ghee, 10kg. of natural honey, a few bottles of expensive wine and many other things. For the remaining things which the family could not provide, they had to compensate by giving money. The "tantrik" then brought a transport and took all the things and left for the cremation ground, while the family members and relatives were instructed to reach the place later at the given time. Once all of them reached there, they found nobody. They were fully deceived. Neither, the cheat nor his helpers could be traced out, even though the patient's two sons were police officials. I felt pity for them and agreed to help them instead.

Thus, in the context of Cancer, I, with all humility, like to mention that its definition, procedure of treatment and role of the people involved in its detection and cure, all have got drastically changed in the last two decades. Now, with so much of development in treatment methods, expert pathologists can detect leukemia, both Hodgkins' and non-Hodgkins'. In many cases MDs in Medicine get updated to detect Cancer with appropriate training and higher vertical education. I had to struggle a lot for understanding these concepts while working hard towards finding out an effective drug that would benefit the entire mankind in near further. My search for an alternative way to the traditional method of treatment of Cancer is still on. I place these explanations because whatever I am saying or doing, in reality

nobody would believe, unless I vouch for the facts. As I have said earlier, the whole spectrum of Cancer treatment in the Indian scenario can never rely just on the medicines but also stand on timely diagnosis, the very line of treatment, affordable drugs, rehabilitation of the family on the patient's terminality, and the like.

With my very little and humble contribution in this field and with an advantage of my location in an urban area, I have honestly and sincerely tried to show that even with meagre resources it is possible to innovate my model or approach towards a wholesome treatment of cancer.

If somewhere the Government thinks that the nucleus of my model should be enlarged I would be happy to serve a million more. I believe: The soldier who fears to fight, the soldier who fears to die, is not a soldier. An ideal soldier always tries to win giving all his might. This is what is important. Winning or losing a battle is not in his hands, but he can always try, and here so I have been trying. In this all-out war against cancer, I consider myself as an ordinary soldier.

The Very Indian Context

In the Indian context, in order to place ourselves on equal footing with the developed nations, it is going to be a very tall process which requires tremendous determination, strong will and faith. The task that we have in our hand at the moment is nearly impossible. The renaissance that benefitted the developed nations back in the 18th century did not have a positive repercussion either on Asia or Africa. As a result only a handful of countries prospered and the rest still miserable. Sometimes this prosperity was at the cost of their colonial subjects. Another reason was the unending love the people of the developed nations had towards their motherland. If we try to get into a detailed analysis we would observe that over the years these nations were moving at a great speed towards the aim of conquering the world market and rule over it. That generation was progressive and dominating by nature. Their love for their country made them think of themselves over and above anyone and this sowed the seed of domination over the others in their mind and soul, and they dominated and ruled over the weak, unskilled and poor population of Asia and Africa. The skill, improved technology and scientific development of the European countries helped them rob the subjugated nations of all their wealth and indigenous industries. Now if I think of my country, the problems we have and the diseases we suffer from, then cancer takes the centre stage. I have again and again stressed on this one point so that the reader who reads only one chapter of this book will be able to relate to the basic problem we are facing right now.

Till now, in India, we have not been able to formulate a proper drug for the healthy cure of a normal headache. Even if one does, it is very important for him to get patent rights and recognition of the global scientific committee.

The right of giving recognition to some research drug remain in the hands of a few influential entities from the developed nations. In order to publish a research paper in a reputed journal needs one to be very well versed with the English language. The parameters that we have set for scaling the educational level of an individual, comes down from the

time of the British rule. When an individual is engaged in some kind of work, he would feel de-motivated to find that in a nation of about 130 crore people no much progress in terms of medical science has been achieved yet. The structure we are currently using is a mix of Colonial legacy and some borrowed infrastructure. This creates an environment of inferiority. He would apparently start questioning the system in totality in due course. The reason being he would ultimately compare our nation with the developed ones in terms of infrastructure, administration, educational liberty, culture and on various other parameters.

At this juncture, what actually we require is a group of experienced doctors and scientists in our country, who would work harmoniously on various drugs available in the market, also engage in testing of food materials and other nutrients and supplements, to analyse their usefulness and requirement in the human body and how our body responds to them in the positive way. The rest that are no longer necessary could be discarded. We need to intake healthy things and this word needs to get spread to everybody. Even over-consumption of nutrients, vitamins or minerals is not healthy. Now-a-days the school-college going students as well as grown-ups are attracted mostly to unhealthy street food, junk food etc. In fact the change in our eating behaviour over the years has contributed towards the weakening of our immune system.

One can resolve his nation's issues only by working in his own country, using its resources, otherwise not. If we think from the point of view of medicine, an Indian working in a developed country has many advantages. Everyone likes to boast about it. But one should also consider the amount of 'brain-drain' that's going on which is affecting our nation in a big way. This is a huge challenge that our country is facing. We have massive human resource at our disposal. The CELL LINE or MICE model do not talk; it is the people facing the situation express their emotions. So one has to perform hair-splitting analysis in order to understand the situation and decide on the steps to be taken next.

What I am trying to say here is that about 3-7% of cancer patients who know me are not coming to seek my help. Compared to the works done specially on lung, liver, gall-bladder, pancreas and stomach cancer through the conventional way, my treatment has proved to be much more effective, and better results have come up in almost every stage. The cost-effectiveness of Psorinum therapy and its non-toxic nature has helped patients to fully recover and to lead a normal healthy life in most of the cases. Our facility, though small, is still equipped with adequate infrastructure, like operation theatre for performing surgeries, a critical care unit and tie-ups with major diagnostic centres and renowned oncologists to visit on weekly basis. Kolkata's eminent oncologists who are associated with us are Dr. Jaydeep Biswas, Director of CNCI (Chittaranjan National Cancer Institute), Dr. Anup Majumdar and Dr. Hiranmay Mukherjee. We have also been recognised by ASCO (American Society of Clinical Oncology) and have published many international papers which are available on the internet.

Again a few patients are there who having felt better after undergoing my treatment, suddenly leave without consulting me even. Some others stop taking the medicine on their whim. Many patients come after suffering from extremity of chemotherapy, but after getting some relief from my treatment go back to take chemotherapy again. Many do not stay in

regular touch also. Most of my patients belong to socio-economically backward class. Desired treatment results are actually obtained from them, but at times it becomes very difficult for me to maintain a database and preserve the medical records of such patients.

I would like to narrate here three such advanced stage pancreatic cancer patients' case-history: Manorama Pati, Jyotsna Karmarkar and Ganesh Chandra Maity.

1. **Smt. Manorama Pati** (Registration No.: 276/10/11, Dated on 28.07.2010): CT scan of her whole abdomen, on 15.07.2010, detected a pancreatic tail mass. The size of the mass was 11.3cm.x11.6cm.x9.2cm. Also lymph nodes were noticed in retroperitoneal region. FNAC (Fine Needle Aspiration Cytology) report on 21.7.2010 showed well differentiated Adenocarcinoma pancreas and minor ascetis.

Without undergoing CT (Chemotherapy), RT (Radiotherapy), or Surgery the patient was admitted to our facility and underwent Psorinum therapy from 28.7.2010. Pancreas appeared compressed in the body and tail region. MDCT (Multiple detector computed tomography) features suggested fairly large well-defined heterogeneously enhancing lesion existing on the left retroperitoneal region with solid necrotic components as well as tiny calcifications within it in the retroperitoneal region. After about one year the patient's CT scan showed that the mass had reduced in size and she was leading a more or less normal life. She could move and eat quite normally. She continued with her tests regularly at three months' interval. Slowly her ascetis also disappeared. On 29.2.2012, CT scan of whole abdomen showed partial regression of size of pancreatic tail SOL (Space-Occupying Lesion). However, the patient did not visit us for further follow up since the end of 2014. The patient expired on 29.05.2015.

Reports and supporting documents of Smt. Manorama Pati.

EKO PATHOLOGY CENTRE



(HISTOPATHOLOGY / CYTOLOGY / FNAC)

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DR. SHYAMALENDU MANDAL

MBBS (CAL) MD (PATH) PGI (CHANDIGARH) MIAC
Ex-SENIOR REGISTRAR - A.I.I.M.S. (NEW DELHI)
CONSULTANT HISTOPATH & CYTOPATHOLOGIST (FNAC)

MS. MONORAMA PATI

Age : 65yrs.

Dt. of receipt : 21.07.10

REFD. BY DR. B. KUMAR

L10892

Dt. of report : 23.07.10

FNAC REPORT (FN/975)

Site of aspiration :

Pancreatic space occupying lesion(SOL)

Gross examination :

CT guided FNAC of pancreatic SOL yielded particulate material.

Microscopic examination :

Smears are cellular. There are sheets and clumps of malignant cells on the background of dense chronic inflammatory cells. The cells have hyperchromatic pleomorphic nuclei with prominent nucleoli, variable cytoplasm and glandular differentiation at places.

Diagnosis:

Well differentiated adenocarcinoma.

Slide enclosed:

DR. SHYAMALENDU MANDAL M.D.
Chief consultant

MRS MONORAMA PATI

65 YEARS

21.07.2010

DR B KUMAR

CT SCAN OF WHOLE ABDOMEN**HISTORY**

Pain abdomen and altered bowel habit.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced MDCT scan of whole abdomen done in the axial plane followed by multiplanar reformations.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no significant abnormality.

Liver appears normal in size, shape, position, outline & density. No focal lesion is detected. The intrahepatic biliary radicles are not dilated. Portal vein appears normal.

Gall bladder appears normal. No evidence of any radio-opaque calculus or intraluminal lesion is detected (However, radio-lucent and small calculus may be missed in CT. USG may be done for further evaluation).

Common bile duct is not dilated.

Pancreas appears compressed in the body and tail regions but is otherwise normal. There is loss of fat planes with pancreatic tail. However, a fairly large well-defined heterogeneously enhancing lesion is seen in the left retroperitoneal region with solid and necrotic components. The lesion measures about 11.3 cm. x 11.0 cm. x 9.2 cm. in size. There is arterial supply to the lesion from the branches of the splenic artery.

Spleen is normal in size, shape and attenuation characteristics.

Both suprarenal glands reveal normal size, morphology and density. No evidence of nodularity or SOL is seen on either side.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal. Both ureters show normal course and calibre.

Contd....2..

MRS M PATI

(CT OF WHOLE ABDOMEN)

21.07.2010

Wall of fully distended urinary bladder is smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Uterus is normal in shape, size and contour. No mass lesion is detected within it. Parametrium is within normal limits.

Both ovaries appear normal. No abnormal mass or cyst is detected in the adnexal regions.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is seen in the peritoneum.

Stomach appears compressed but is otherwise normal. The bowel loops are unremarkable.

Bones under review show degenerative changes in the visualized vertebral bodies. Parietal and paravertebral muscles including psoas muscles are normal.

IMPRESSION

MDCT features suggest a well-defined fairly large heterogeneously enhancing soft tissue mass with solid and necrotic components as well as tiny calcifications within it in the retroperitoneal region causing compression to the surrounding structures. However, there is loss of fat planes with the pancreatic tail.

Features favour a neoplastic condition - ? pancreatic tail neoplasm vs. ? retroperitoneal mass vs. any other pathology.

Suggested clinico-pathological correlation and other investigations for further evaluation and confirmation if clinically indicated.


DR S K SHARMA
MD
DR S ROY
MD
DR S KUNDU
DMRDDR S KUNDU
MD

DRS. TRIBEDI & ROY
DIAGNOSTIC LABORATORY
NABL ACCREDITED (ISO 15189 : 2007)



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Phones : 2226-6643 / 8789 / 5961
E-mail : roylab@vsnl.net

BRANCH : 48A, Diamond Harbour Road,
Kolkata-700 027, (9 A.M. - 3 P.M.)

Certificate No. : M0035

NAME Manorama Pati 65 yrs. (Lab No. DKT 887)
ADDRESS _____ DATE OF RECEIPT 24.07.2010
PHYSICIAN Dr. B. Kumar DATE OF REPORT 27.07.2010
MATERIAL FNAC slides for review

FNAC SLIDES FOR REVIEW

Received two stained slides No. F – 975 of FNAC from Pancreas.

REPORT :-

Smears are cellular and show clusters as well as scattered malignant cells. These cells have hyperchromatic nuclei with irregular nuclear membrane, prominent nucleoli and variable amount of cytoplasm.

DIAGNOSIS :- Pancreatic SOL ----- Well differentiated
Adenocarcinoma.

ADVICE :- Clinical correlation.

F/2266/10

Smear - 02

DR. SAYED. M. NADEEM M.D

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MANORAMA PATI

65 YRS

14.08.2011

DR. OF MCH

26033/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient of Ca.tail of pancreas – last CT 20.01.11.

TECHNIQUE

Plain, oral and I.V. (non –ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no obvious abnormality.

Liver is normal in size, shape, position, outline and density. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Porta hepatis appears normal.

Gall bladder appears normal and its lumen does not show any radio-opaque calculus or intraluminal lesion (However, radio-lucent and small calculus may be missed in CT. USG may be done for further evaluation).

Common bile duct is not dilated.

Pancreas : A variagated mass seen at tail of pancreas measuring 100mm x 96mm. Head & body are normal.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

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-2-

MANORAMA PATI

65 YRS

14.08.2011

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Uterus is normal at per age.

Adnexal regions are clear.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

Lung bases are clear.

IMPRESSION

Review study shows almost identical radiological appearance of pancreatic tail mass since last study of 20.01.11. (100mm x 96mm on 14.08.11, 110mm x 93mm on 20.01.11.)

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD


DR.ANUP SADHU
DMRD, MD.

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MONARAMA PATI

66 YRS

29.02.2012

005

DR.OF MCH

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient of ? pancreatic tail neoplasm – last CT 21/7/10.

TECHNIQUE

Plain, oral and I.V. (non –ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no obvious abnormality.

Liver is normal in size, shape, position, outline and density. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Porta hepatis appears normal.

Gall bladder appears normal and its lumen does not show any radio-opaque calculus or intraluminal lesion (However, radio-lucent and small calculus may be missed in CT. USG may be done for further evaluation).

Common bile duct is not dilated.

Pancreas – Head & body are normal. Fairly large variagated mass seen in the tail (105 x 105 mm)

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

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-2-

MONARAMA PATI

66 YRS

29.02.2012

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Uterus is normal in shape, size and contour. There is no mass lesion in it. Parametrium is within normal limits.

Adnexal regions are clear.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

IMPRESSION

Review study shows partial regression of size of pancreatic tail S.O.L. since last study of 21/7/10.
(21/7/10 – 113 x 110 mm, 29/2/12 – 105 x 105 mm)

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD


DR.ANUP SADHU
DMRD, MD.

2

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005

MONARAMA PATI

66 YRS

29.02.2012

DR.OF MCH

C T SCAN OF THE BRAIN

TECHNIQUE

Plain & I.V. (non-ionic) contrast enhanced C T scan of the brain done with 5 mm. and 10 mm. sections in the axial plane.

FINDINGS

Posterior fossa shows prominent cerebellar folia on both sides. Fourth ventricle is in the midline. Basal subarachnoid cisterns, sylvian fissures and the cortical sulci are widened. Third and both lateral ventricles are mildly dilated with septum in the midline.

No focal lesion is detected in the brain parenchyma.

IMPRESSION

Diffuse brain parenchymal shrinkage.

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD


DR.ANUP SADHU
DMRD, MD.

(8)

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তারিখ	চিকিৎসার বিবরণ	তারিখ	চিকিৎসার বিবরণ
	<p>(২)</p> <p>known case of Pancreatic Tail Adenocarcinoma diagnosed in 07/10. No treatment received. Now on reassessment the tumor hasn't increased in size markedly. R @ minimally symptomatic. </p>		<p>(৩)</p> <p><u>Adx</u> = On view of the proved nature of the tumor and the being no evidence of spread although may be made at resection provided size is far from the metastatic point of view.</p> <p>Any TTP & send 1. m. x. m. T2 after lunch</p> <p>Lab, R, D, E, N Super TTP Urea, Creatinine BUN, Platelet count LFT, PT time HIV, HbsAg, HbsAb Chest xray, PFT ECG Echocardiography</p> 

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MANORAMA PATI

66 YRS

22.11.2012

DR. OF MCH

18977/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient of Ca.tail of pancreas – last CT 14.08.11.

TECHNIQUE

Plain, oral and I.V. (non –ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no obvious abnormality.

Liver is normal in size, shape, position, outline and density. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Porta hepatis appears normal.

Gall bladder appears normal and its lumen does not show any radio-opaque calculus or intraluminal lesion (However, radio-lucent and small calculus may be missed in CT. USG may be done for further evaluation).

Common bile duct is not dilated.

Pancreas – Fairly large variagated mass (99mm x 94mm) seen in pancreatic tail.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

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-2-

MANORAMA PATI

66 YRS

22.11.2012

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Uterus is normal in shape, size and contour. There is no mass lesion in it. Parametrium is within normal limits.

Adnexal regions are clear.

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

Lung bases are clear.

IMPRESSION

Review study shows marginal regression of pancreatic tail mass since last study of 14.08.11 (On 22.11.12 – 99mm x 94mm, On 14.08.11 – 100mm x 96mm)

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD

DR.ANUP SADHU
DMRD, MD.

2. **Jyotsna Karmakar**, a 56 old lady, was admitted at the Gastroenterology department of SSKM Hospital, Kokata following severe abdominal pain. On 16.4.2012 a CT Scan was conducted of the whole abdomen. CT features suggested mild hepatomegaly with pnuemobilia, decreased attenuating lesion in the left lobe and pancreatic head SOL. FNAC dt. 23.05.2012 from pancreatic mass suggested malignant epithelial lesion Adenocarcinoma (primary/secondary deposit). On 28.5.2012 stenting was done. From 26.7.12 Psorinum Therapy was started. Gradually the patient's condition improved. She was asked to undergo a CT Scan, but she refused. The patient till date is leading a healthy life.

Reports and supporting documents of Jyotsna Karmakar.

ANJAY BANERJEE
M.D., D.M.S.
Assistant Professor
Medical Gastroenterology
Col of Dist. & Subdiv. Liver Diseases
GME & R. SSKM Hospital, KOL-20

DEPARTMENT OF HEALTH & FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
OPD Patient Card
S.S.K.M. HOSPITAL & IPGME & R
A.J.C. ROSE ROAD, KOLKATA-20

✓

Paid Rupees 2.00

Name : JYOTSNA KARMAKAR	Yrs. 55	Month 20	Days 08	Day : Wednesday	Reg. No. 8618428228
Sex : Male	Age 55	Yrs. 20	Month 08	Days 08	Reg. Date 28-Oct-2018
Ref. From :					Card No. Q218373927/1
Visit No. : 1	Department : Gastroenterology OPD.	Visit Date : 28-Oct-2018	Time : 09:54	Entry No. :	
Doctor/Unit Name (DOW) : 00C00000555 - Wednesday	Room No. 21:				

Visit No. : 2	Visit No. : 3	Visit No. : 4
Visit Date : Tm.	Visit Date : Tm.	Visit Date : Tm.
Department :	Department :	Department :
Doctor/Unit :	Doctor/Unit :	Doctor/Unit :
Entry No. :	Entry No. :	Entry No. :

Clinical Notes	ADVICE
Post-renal x (kg ym trace) ↓ now pain abdomen x 3m low grade fever (?) no icteric vomiting. w. lay w. chn w. —	Ad. ER ct on 23/10/10 13hr P time (prothrombin time) Rw. f platelet count Etal will be done if then report normal SR 20/10/10

22/24

DEPARTMENT OF HEALTH & FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
OPD Patient Card

23 FEB 2011

Clinical Notes

ADVICE

? cholelithiasis

Had jaundice & chills

Post-cholec

gone ERCP in Oct '10

MRCP (Feb '11) - definite evidence
of bile duct

Cholelithiasis

Adh

✓ USG whole abdomen (transverse)

• called Gastroenterology OPD

SP
23/2/11

Refer to Dr. Sukanta B., Thursday

Gastroenterology OPD (RN-4)

SP
23/2/11



Suraksha

AROGYAM ANANDAM

Polyclinic Diagnostics Pharmacy

Patient Code : KHA/18-02-2011/235458 Patient Id : P/392010
Patient Name : JYOTSHNA KARMAKAR
Age : 55 Years Sex : Female
Referred Doctor : DR. SANTANU DAS
Date of Test : 18/02/2011
Report printed on : February 19, 2011

DEPARTMENT OF MRI

REPORT ON EXAMINATION OF MRCP

Clinical information: Abdominal pain

Protocol:

MRI of the upper abdomen was performed in axial & coronal planes using T1 and T2 weighted fast spin echo. Projectional MRCP images were obtained using 3D TOF.

Observations:

- Intrahepatic biliary radicles are dilated. Right and left hepatic ducts appear dilated. There is an abnormal well-defined lesion of mixed echogenicity, predominantly hypointense on T1W and T2W, noted surrounding and compressing the common bile duct at porta hepatis. There is evidence of pneumobilia.
- Gall bladder is not visualized (--? Post-operative).
- Main pancreatic duct is not dilated.
- Liver, spleen & pancreas are normal in size, outline & signal intensity.

Conclusion:

- Abnormal lesion surrounding and compressing the common bile duct at porta hepatis causing intrahepatic biliary radical dilatation.

Advice: Further investigations.
Clinical correlation.

DR. MOUSUMI ROYCHOWDHURY (SAHA)
MD, RADIO DIAGNOSIS

V.Id.:D02-194 ULT-16
Patient Name :Mrs. Jyotshna Karmakar
Age:52years , Sex:Female
Referred by Dr. Santanu Das. MBBS, DIP CARD

Booking Date :03/04/12

Reporting Date :03/04/12

USG REPORT OF WHOLE ABDOMEN

LIVER :

Very coarse texture with dilated intrahepatic biliary ducts. Small echogenic material seen in side intra hepatic biliary channels. Multiple hypodense liver opacities. Size at mid clavicular plane -- 98.4 mm.

GALL BLADDER :

Not seen.

C.B.D. :

35.1 mm. in diameter. Gross dilated & occupied by strong echogenic material & stent (cholangio Ca vs. thick sludge).

PORTAL VEIN :

8.3 mm. in diameter.

PANCREAS :

Normal in size, shape and echotexture. No obvious focal lesion or intraparenchymal calcification seen. Main pancreatic duct is not dilated. No peripancreatic fluid collection seen.

SPLEEN :

Spleen is normal in size (76.2 mm. in long axis) shape and echotexture. No focal lesion seen.

KIDNEYS :

Both the kidneys are normal in size, shape and axis. Cortical echotexture and cortico-medullary differentiation are normal in both sides. No evidence of any focal lesion seen in either kidneys. No hydronephrosis detected.

Right kidney measures : 102.8 mm.

Left kidney measures : 104.5 mm.

URETERS :

Pelvi-ureteric junction and vesico-ureteric junctions are normal. No obvious intraluminal lesion seen in visible part.

Contd...P/2.

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Building World Of Accuracy

(2)

V.Id.:D02-194 ULT-16
Patient Name :Mrs. Jyotshna Karmakar
Age:52years , Sex:Female
Referred by Dr. Santanu Das. MBBS, DIP CARD

Booking Date :03/04/12

Reporting Date :03/04/12

URINARY BLADDER :

Optimally distended, normal in shape and wall thickness. No evidence of any intraluminal lesion seen.

UTERUS

Postmenopausal. Endometrial cavity is clear. No solid SOL noted. Cervix looks normal.

OVARIES :

Not seen.

No ascites / para-aortic lymphadenopathy noted.

IMPRESSION

: Gross dilated C.B.D. with stent (cholangio Ca vs. thick sludge).

-- Please correlate clinically.

DR. RANJAN SUKLA GHOSH
MBBS, DMRD (Cal)
Consultant Radiologist.

ACCURATE MEDICARE & DIAGNOSTIC PVT. LTD.

21, B. T. Road, Rabindra Bhaban, Khardaha, Kolkata - 700 117, ☎ : 2583 7780; 2563 9111, e-mail : accurate_medicare@yahoo.com

Building World Of Accuracy

EKO CT & MRI SCAN CENTRE

A UNIT OF EKO DIAGNOSTIC PVT. LTD.

(A Joint Venture with Eko Diagnostic Pvt. Ltd. &
Dept. of Health & Family Welfare, Govt. of West Bengal)



JYOTSNA KARMAKAR

56 YRS

16.04.2012

DR OF MCH

46367/018

CT SCAN OF UPPER ABDOMEN

HISTORY

? Hepatic metastasis.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced CT scan of upper abdomen done with 5 mm and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the upper abdomen in supine position and in frontal projection shows no significant abnormality.

✓ Liver is enlarged with pneumobilia. Low density lesion seen in left lobe.

✓ Gall bladder is removed.

✓ Common bile duct is dilated upto lower end.

✓ Pancreas : Head is bulky.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd..2..

& MRI SCAN CENTRE

OF EKO DIAGNOSTIC PVT. LTD.

Joint Venture with Eko Diagnostic Pvt. Ltd. &
(Dept. of Health & Family Welfare, Govt. of West Bengal)



-2-

JYOTSNA KARMAKAR

56 YRS

16.04.2012

Aorta and IVC are normal. No sizeable para-aortic, mesenteric or retroperitoneal lymph node is detected. No free fluid is detected in the peritoneum.

Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

IMPRESSION

CT features are suggestive of mild hepatomegaly with pneumobilia, decreased attenuating lesion in left lobe & ? pancreatic head S.O.L.

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD


DR.ANUP SADHU
DMRD, MD.

V.Id.:E18-188 PAT-2
Patient Name :Ms. Jyotshna Karmakar
Age:55years , Sex:Female
Referred by :

Booking Date :23/05/12

Reporting Date :23/05/12

USG GUIDED FNAC FROM PANCREATIC MASS

Slide No. : AMD-F69/12

Clinical Data : Cholecystectomy (2002), developed CBD obstruction with mass in head pancreas which is sampled by guided FNAC.
Smears received from imaging division.
USG – hepatic metastasis suspected also.

Cytology : Smears are haemorrhagic and cellular consisting of cell aggregates of epithelial cells with moderate anisonucleosis of the cells.
Nucleoli are prominent.

Impression : Malignant epithelial lesion – ? adenocarcinoma (primary/secondary deposit).

HP is solicited for final diagnosis.

Dr. Md. Sawkat Ali
MBBS, DCP, MD (Path)
(Consultant Cytopathologist & Histopathologist)

Dr. P. K. Sarkar
MBBS, DCP

Dr. Sandip Chak
MBBS, MD (Bioc)

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3. **Ganesh Chandra Maity** is a 60 year old poor field-labour. He was admitted at this facility on 3.10.2014. The CT Scan conducted on 29.9.2014 had shown impression of biliary stricture (likely neoplastic) at the level of RHD (right hepatic duct) & LHD (left hepatic duct) confluence with secondary biliary obstruction, upper retroperitoneal lymphadenopathy, minimal traces of ascites, bilateral pleural effusion with atelectasis of basal lung segment and jaundice. MRCP (Magnetic resonance cholangiopancreatography) done on 30.9.2014 showed impression of obstruction to the biliary tree at porta hepatis with isolation of right and left sided hepatic ducts. Incidentally consolidation was seen in lower lobe of both lungs with bilateral pleural effusion. Malignant cells of ascites were also found.

The patient was admitted here for one and a half months and Psorinum Therapy was administered from the very next day of his admission. Since the patient's condition was improving at a good pace and due to his financial weakness, his treatment was made absolutely free. On 25.9.2015 a CT Scan was conducted, which suggested mild hepatomegaly with pnuemobilia and a few adhered gut loops at lower abdomen. The patient is absolutely fine now and leading a normal life.

Reports and supporting documents of Ganesh Chandra Maity.

 RUKSHI <i>Diagnostic Centre</i> DAHARPUR TAMLUK PURBA MEDINIPUR (T) 973 (03226) 267207, Mobile: 9436230498, 9434417310	
With best complements to DR. P. JANA, MD(CAL) Name: GANESH MAITY Age: 55 Yrs Sex: M Date: 29.09.14 Part of exam: USG OF WHOLE ABDOMEN	
<u>LIVER:</u> Is enlarged in size. Parenchymal echotexture is altered at the porta. Intrahepatic biliary radicals are dilated. Portal vein and (8mm) hepatic veins are of normal caliber and distribution. Minimal perihepatic fluid collection noted	<u>URINARY BLADDER:</u> Wall is smooth & regular. (catheter seen in situ) <u>PROSTATE GLAND:</u> Not visualized
<u>GALL BLADDER:</u> Is contracted <u>COMMON BILE DUCT:</u> Not dilated (3 mm). No intraluminal pathology seen. <u>PANCREAS:</u> Normal in size, contour outline and parenchymal echogenicity. No focal or diffuse parenchymal change seen. Main pancreatic duct is not dilated. <u>SPLEEN:</u> Is enlarged (114mm) in size. No focal or diffuse parenchymal lesion is identified. <u>KIDNEYS:</u> Both kidneys are normal in size, shape, parenchymal echogenicity and cortical thickness. No focal SOL, calculus or hydronephrotic change is noted. RK measuring 95mm LK measuring 96mm <u>URETERS:</u> Ureters are not dilated.	** Minimal right sided pleural effusion seen <u>IMPRESSION:</u> 1. Hepatomegaly with dilated IBHRs & altered parenchymal echotexture at portal region. CBD is of normal caliber -? Obstruction at porta (Bifurcation of hepatic ducts) - ?? Neoplastic etiology. 2. Minimal perihepatic fluid collection 3. Splenomegaly <i>Clinical correlation & other investigations (CT scan abdomen) suggested.</i>  DR. PRITAM DAS, MD (RADIOLOGISTS) CONSULTANT RADIOLOGIST
ECHOCARDIOGRAPHY (COLOUR DOPPLER) • TVS • USG OF UPPER & LOWER LIMBS THYROID USG OF COLOUR DOPPLER DONE HERE	



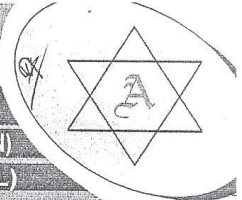
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SREE AUROBINDO

DIAGNOSTIC & ANALYTICAL CENTRE

SCANNING CENTRE PVT. LTD. (WHOLE BODY SPIRAL CT SCAN)

X RAY (500mA DIGITAL)



SANKARARA BUSPOOL • TAMLUK • PURBA MEDINIPUR

Thanks for referral

With best compliments to DR. P. JANA MD (CAL)		
Name: GANESH MAITY		
Age: 53 YEARS	Sex: M	Date: 29/Sep/14
Part of exam: CT SCAN OF WHOLE ABDOMEN (PLAIN+CONTRAST)		

Scans are taken in axial section through the abdomen from the top of liver above to symphysis pubis below with 5 mm slices at 5 mm table steps with oral + rectal contrast without and with I. V. contrast.

LIVER: Is marginally enlarged in size (16 cm in MCL). There is no cyst / abscess / mass seen in liver. Hepatic venous system is normal. Hepatic parenchymal attenuation & enhancement pattern is normal in all the lobes of liver.

GALL BLADDER & BILIARY TREE: GB is grossly contracted with thickened wall. Bilobar IHBRs are marginally dilated with non-visualisation of LHD & RHD confluence. LHD & RHD measure 07 mm & 06 mm respectively. Proximal part of CBD is prominent & distal part is collapsed. Hyperdense sludge noted in dilated IHBRs & proximal part of CBD.

PANCREAS: Exhibits normal size, shape & position. Pancreatic parenchymal attenuation pattern is normal. There is no evidence of any focal mass or peri-pancreatic collection. MPD is not dilated. Parenchymal enhancement pattern is normal.

SPLEEN: Is normal in size, shape & position. No focal mass / abscess seen. Parenchymal enhancement pattern is normal.

KIDNEYS: Both the kidneys show normal size, shape, position & configuration. Corticomedullary differentiation is maintained. No mass / hydronephrosis / calculus / cyst seen on either side. Parenchymal enhancement pattern is normal. Excretion of contrast seen bilaterally.

URETERS: Both ureters in visible portions are normal in caliber, course & configuration. No sign of obstruction seen.

Contd.....P/2.....

Asomal
DR. ANINDYA SASMAL

MD (RADIOLOGY), (IPGMR - CAL.)
CONSULTANT RADIOLOGIST & SONOLOGIST

DR. SUMERU CHAKRABORTY

MBBS (CAL), DMRD (RADIOLOGY) (BANG)
CONSULTANT RADIOLOGIST & SONOLOGIST

DR. SUBHASIS GOSWAMI

MD (RADIOLOGY), (IPGMR - CAL.)
CONSULTANT RADIOLOGIST & SONOLOGIST

This is a Professional opinion only & not the diagnosis. It should be clinically correlated. Patient's identification not verified.

REPORT IS NOT VALID FOR MEDICOLEGAL PURPOSE.

OUR SERVICE → 2D,3D & 4D ULTRASONOGRAPHY • COLOUR DOPPLER • ECHOCARDIOGRAPHY • DIGITAL X-RAY • SPIRAL CT

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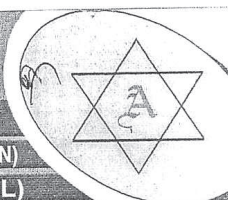


SREE AUROBINDO

DIAGNOSTIC & ANALYTICAL CENTRE

SCANNING CENTRE PVT. LTD. (WHOLE BODY SPIRAL CT SCAN)

X RAY (500mA DIGITAL)



SANKARARA BUSPOOL • TAMLUK • PURBA MEDINIPUR

Thanks for referral

Page No -- 2

Patient's name: GANESH MAITY 53 YEARS / Male

URINARY BLADDER: Is well distended. **Wall is thickened.** No sign of stone / mass noted.
Foley's catheter bulb seen in situ.

PROSTATE: Is normal in size, shape, configuration & attenuation. No mass is seen.

STOMACH: Shows normal size, shape & configuration. No sign of mass / diverticulum seen. No obstruction is also observed.

DUODENUM: Shows normal size, shape & configuration. No sign of mass / diverticulum / obstruction in visible portions.

JEJUNUM & ILEUM: Shows normal loops having normal mucosal pattern. No sign of mass / diverticulum / obstruction noted.

ILEO-CAECAL REGION: Exhibits normal study with no sign of mass / obstruction.

LARGE INTESTINE: Caecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon & rectum are normal. No sign of mass / obstruction seen.

** Minimal traces of ascites, bilateral pleural effusion and atelectasis of basal lung segments noted.

** Few discrete & conglomerated periportal, paracaval and para-aortic lymph nodes are seen, largest one measures 1.6 cm x 1.3 cm.

IMPRESSION:

- Biliary stricture (likely neoplastic) at the level of RHD & LHD confluence with secondary biliary obstruction, upper retroperitoneal lymphadenopathy, minimal traces of ascites and bilateral pleural effusion with atelectasis of basal lung segments
--- relevant further investigations like MRCP / ERCP suggested.
- Cystitis.

Please correlate clinically.

ASASMA
DR. ANINDYA SASMAL
MD (RADIOLOGISTS), (IPGMR - CAL.)
CONSULTANT RADIOLOGIST & SONOLOGIST

DR. SUMERU CHAKRABORTY
MBBS (CAL), DMRD (RADIOLOGISTS) (BANG)
CONSULTANT RADIOLOGIST & SONOLOGIST

DR. SUBHASIS GOSWAMI
MD (RADIOLOGISTS), (IPGMR - CAL.)
CONSULTANT RADIOLOGIST & SONOLOGIST

This is a Professional opinion only & not the diagnosis. It should be clinically correlated. Patient's identification not verified.
REPORT IS NOT VALID FOR MEDICOLEGAL PURPOSE.

OUR SERVICE: 2D/3D & 4D ULTRASONOGRAPHY, HECOCARDIOGRAPHY, DIGITAL X-RAY, SPIRAL CT

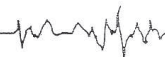


"JAI MATA JI"

Lakhotia

Diagnostic Service Pvt. Ltd.

Regd. Office : 493/C/A, G.T. Road, Vivek Vihar, Block - E, Howrah - 711 102



REPORT

PT'S NAME : GANESH CHANDRA MAITY
REF. BY : DR. of P M TREAT PVT LTD
SEX : MALE / AGE : 55 YRS.

CODE : 30.09.6060
DATE OF INV. : 30.09.2014
DATE OF REP. : 30.09.2014

Thank you for referring the patient for Scan.

M. R. C. P. ✓

HISTORY : Pain.

TECHNIQUE : MRCP images taken in various rotations followed by axial & coronal T1 and T2 weighted sequences.

FINDINGS : Intrahepatic biliary radicles are minimally dilated. Confluence of right and left hepatic ducts is not seen. Common bile duct is not seen. No calculi seen.

Gall bladder is not seen.

Pancreatic duct is normally seen.

Screening of upper abdomen reveals :

Liver & : Gall bladder is not seen. Multiple vessels are seen at the porta hepatis.

Gall Bladder : Portal vein is normally seen.

Pancreas : It reveals normal signal characteristics and contour.

IMPRESSION : Obstruction to the biliary tree at porta hepatis with isolation of right and left sided hepatic ducts.

Incidentally consolidation is seen in lower lobe of both lungs with bilateral pleural effusion.

DR. BANIBRATA MAZUMDAR
MD. (RADIODIAGNOSIS)
CONSULTANT RADIOLOGIST

KB

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GANESH CH. MAITY

55 YRS

25.09.2015

DR. OF MCH

12042/018

C.T.SCAN OF UPPER ABDOMEN**HISTORY**

Pain abdomen.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced CT scan of upper abdomen done with 5 mm and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the upper abdomen in supine position and in frontal projection shows no significant abnormality.

Liver is enlarged with pneumobilia.

Gall bladder is not optimally delineated.

Common bile duct is not dilated.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd..2..

AT Medical College & Hospital Campus

88, College Street, Kolkata - 700 073, Phone : 2212-3778/3779

Identity of the patient not verified, If there is any lack of correlation between the result and clinical condition, please refer the patient to the respective department.

-2-

GANESH CH. MAITY

55 YRS

25.09.2015

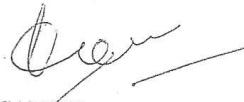
Few distended gut loops seen.

IMPRESSION

CT features are suggestive of :

- 1) Mild hepatomegaly with pneumobilia - ? Cause.
- 2) Few adhered gut loops at upper abdomen.

Suggested clinical correlation and further investigations if clinically indicated.

DR.D.SHARMA
MDDR T.K.DHAR
MD (Radiodiagnosis)
DR.ANUP SADHU
DMRD, MD.

On The Relevance Of My Therapy In The Indian Context

When I decided to become a part of the medical community, I had no one by my side to guide me on what to study and when to study. I had no pre-defined syllabus with me. So I alone had to decide upon what to know and who would be the right person to approach for the detection and treatment of cancer. I had to learn each and every intricate detail in my journey so far. I would have liked to explain these things in detail, but due to several constraints I refrain from putting all my thoughts in this book. As I have already mentioned, NGOs played a very crucial role in my journey so far. I have always tried that even if I discontinue my association with these NGOs due to some reason, I must make sure that the objectives it meant to serve reach the ultimate goal. Unfortunately, except for Oncolink, no other NGO could sustain, and at that juncture Rabindranath Chatterjee Cancer Memorial Trust came actively forward.

When we started with some new work the question that arose is who would be the people to authenticate our clinical trials and other scientific works. In my journey I have learnt that pathology and radiology form the core of cancer diagnosis. In the first step, pathology characterizes the specific histologic and molecular features of tissues, while radiology localizes suspicious lesions and informs about clinical-stage and potential co-morbidity determinations.

In the next step while the radiologists provide a diagnostic imaging service to patients, the oncologists are medical specialists who are skilled in the treatment of cancer using radiotherapy, chemotherapy, hormone therapy, radioactive isotopes and other specialised techniques. In addition to treating those patients who are subsequently cured of their disease, the oncologist is normally the only physician to manage the patient through the whole course of the patient's cancer treatment, along with general local physicians. Clinically to what extent the patient's medical condition has improved would be determined jointly by the oncologist and the radiologist.

There were no committees in India at least 25 years ago which could authenticate such new scientific works. I had to create two such committees with some eminent personalities in the field of Cancer, and these committees had to break-through typical stereotype of the prevailing socioeconomic barriers and had to overcome many uneven situations in due course. I had to convince a group of pathologists to do a scientific work under the leadership of Prof. Subir Dutta to show the joint role of pathologists and radiologists in the detection of cancer. Another committee consisting of Oncologists and Radiologists looked into the aspect of their joint responsibilities in treating cancer and to check to what extent the patient had been cured and the tumour had regressed without the application of radiotherapy and chemotherapy, or not.

To avoid any legal troubles we chose to work with patients who were resistant to chemotherapy and radiotherapy. We also worked with advanced-stage patients on whom chemotherapy and radiotherapy cannot be administered and also surgery cannot be performed, like patients having cancer in lung, liver, stomach, gall-bladder and pancreas, and also the socio-economically backward patients. This I learnt when I had worked at Cure Point

Nursing Home under the guidance of Prof. R. N. Brahmachari and Prof. Anup Majumdar. In subsequent stages I tried to pen down these case-scenarios as they came on my way and presented this model of treatment in the Rajasthan session of Science Congress.

Since it was a new work, there lay two questions before me to be answered. Firstly, why I got involved in a different mode of treatment that did not follow the ongoing conventional methodology? Secondly, to show the efficacy and progress of the treatment I had very limited time in hand which was definitely inadequate. How to react to this challenge? My published paper on this subject throws light on all these aspects and answers many questions.

Some questions always haunt me. Firstly, who am I? Why am I doing this? Why an unknown person working with an unknown material determines to make this world Cancer free? Secondly, it has to be proved beyond doubt whether the material used to prepare the drug is toxic or not. Thirdly, why conventional treatment was not adopted? In the published paper, viz. "Non-conventional Treatment of Tobacco Related Cancer Gradually gets Right Perspective through Psorinum Therapy", I have tried to explain some of these issues and replies thereto. As a result, the people who wanted to check me not only got convinced in due course but also many of them got associated with us. I have worked on quite a few papers in collaboration with them, which have been certified nationally as well as globally.

If somewhere in the near future my contribution in this field be ever evaluated, then it will be seen that the infrastructure needed has also been innovated by me.

While going through my work, it may appear to one as a game of soccer as to how I have held the ball beneath my feet and how I have scored the goal. The referees would form a group to decide whether the goal is to be awarded to my credit or mark it as a foul. Before acknowledging my work I would urge the scientific world to consider my position as reflected herein.

As I have earlier mentioned that my primary target was treating those patients who were getting treated at SSKM Hospital and Chittaranjan National Cancer Institute (CNCI) so that I can show how they are responding to my treatment and benefitted, and this too gets documented in the hospitals' as well as the patients' medical records. In order to do so, on many occasions I had to go out of the way and literally win away a few patients of the concerned institutes to proceed with my work. I was actually trying to supplement the works of those institutes and at the same time to prove myself to them. In this context I would like to remind the reader about a patient named S. P. Deb Roy who was earlier getting treated at SSKM Hospital. A few other advanced colon cancer patients had in the meanwhile died. This was primarily due to lack of infrastructure and knowledge-deficiency. But a very advanced colon cancer patient, S. P. Deb Roy, got absolutely cured under my treatment. From Chittaranjan National Cancer Hospital the two patients whom I was treating were Smt. Parul Bala Dey and Bijoy Kumar Hui. Smt. Parul Bala Dey was an advanced CA lung patient and Sri. Bijoy Kumar Hui was an advanced CA stomach patient.

SMT. PARULBALA DEV,
Lung C.A.

Smt. Parul Bala Dev 75 yrs. Hindu widow now residing at Bidhan Colony, Patipukur, Calcutta - 700 048 presented with cough with foetid expectoration, anorexia and vomiting.

X-ray chest PA dated 29.11.90 shows homogeneous opacity in Rt. upper zone suggesting Rt. upper lobe consolidation. TC-11, 8.00 DC-normal, sputum was negative for AFB and malignant cell.

Ampicillin 500 mgm three times daily for 7 days, vitamins and expectorents was prescribed.

Repeat X-ray chest PA dated 18.01.91 shows homogeneous opacity in Rt. upper zone with shifting of Trachea to Rt. and raised Rt. dom of diaphragm suggesting collapse consolidation of Rt. upper lobe. Rt. Lat view dated 28.01.91 also support collapse consolidation. There was a possibility of pressure on Rt. upper lobe bronchus by a mass.

At this stage the patient is referred to Chittaranjan National Cancer Institute for further investigation and treatment. The patient attended CRNCI on 23.01.91 and advised X-ray chest Rt. Lat view. Sputum for AFB and malignant cell, Cap. Tetracycline (Oracip) 500 mgm twice daily for 7 days and on 29.01.91 Tab Bactrim DS twice daily for 5 days. Sputum cytology for malignant cell and for AFB was negative, sputum culture for tubercular bacillus was negative. On 14.02.91 they decided to start Radiotherapy and the patient was advised to report on 25.02.91 for 1st. dose of Radiotherapy.

Contd....

But the patient party decided not to give Radiotherapy. They brought the patient to The New Resource on 23.02.91 and Homeopathy treatment started by Dr. Asim Kumar Chatterjee under the guidance of prof. R. S. Bhakta, Presidency Surgeon, National Medical College. After treatment for a month cough was relieved completely and there was improvement of general health. X-ray chest dated 16.04.91, 16.05.91 and 05.07.91 shows radiological improvement with regression of lesion. TC-7500, Hb-9.7 gm.

After this the patient discontinued the treatment for some time and again reported to this Institute on 07.09.91. X-ray chest PA dated 12.09.91 and lateral view dated 14.09.91 shows much extension of lesion. Homogeneous opacity in Rt. upper zone suggestive of consolidation of Rt. upper lobe. Patient was suffering from cough and dyspnoea. Bronchoscopy was done by Dr. Abani Biswas (Thoracic Surgeon, National Medical College) on 15.09.91 but due to profuse bleeding nothing could be visualised inside. X-ray dated 16.09.91 shows some extension of the lesion which may be due to deposition of blood in alveoli and in broncheal tree.

Hemeopathy treatment was started again from 17.09.91. X-ray chest dated 05.11.91 and 20.11.91 shows further increase of the lesion but there was some improvement of general health. X-ray dated 21.12.91 shows partial resolution of the lesion and X-ray chest PA and Lateral dated 19.02.92 shows much regression of the lesion. There was improvement of the general health of the patient.

It appears from the above findings that when this homeopathic medicine was applied from last week

Contd....

of February '91 to the middle of July '91 the patient responded well the symptoms releived, general health improved and radiologically the lesion regressed. The diseased deteriorated when the treatment is discontinued from middle of July '91 to last week of August '91 and again there is physical and radiological improvement when the medicine started again from first week of September '91.

Regarding the diagnosis of the disease there may be some doubt. From the beginning tuberculer basilli could not be isolated and the patient not responded to several course of antibiotic. So diagnosis of lung cancer is asumed and treated in this line.

Patient is still in good health and leading a normal life.



Dr. ASIM KUMAR CHATTERJEE

D.M.S. C.M.L

PRESIDENT, THE NEW RESOURCE

Report on the Examination of Blood

Name Sh. Parul Bala Dey. Age 72 Years.
Sent by Dr. _____ On 29.11.71

Hæmoglobin

100 P c 14 gram (Sahli)

Enumeration of Blood Corpuscles :

Red _____ per c mm
White 11,400 per c mm

Differential Count :

Neutrophils	68	per cent
Lymphocytes	25	per cent
Monocytes	00	per cent
Eosinophils	07	per cent
Basophils	00	per cent

Abnormal Red Cells or White cells :

Mycelocytes	nil.
Poikilocytosis	nil.
Anisocytosis	nil.
Polychromatophilia	nil.
Reticulocytes	nil.

Special Test :

Parasites :

Malaria Parasite

Microfilaria

Other Examination

Formaldehyde test

(for kala azar)

Stibaniline Test (")

Coagulation mts sec

Bleeding time mts sec

Sedimentation Rate

(Westergren method)

1st hour 140 h.h.

2nd hour

Mean

Agglutination Reaction against :

T. H.

T. O.

Para A

Para B



Usha X-Ray & Diagnostic Clinic
Subhas Avenue, Ranaghat, (Nadia)
Phone :- R G H 274

Parul Bala Dey
Signature

Working Hours 7 a. m. to 1 p. m.
3 p. m. to 8 p. m.

Dial.: { 55-8522
54-4000

Dr. M. C. Paul's Bacteriological Laboratories

131C, BIDHAN SARANI, CALCUTTA-700 004

(Near Shyambazar Tram Depot)

LABORATORY EXAMINATION REPORT

Date of Receipt 8.12.90

Date of Report 25.1.91

Ref. No. 43

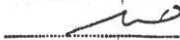
Patient's Name Parulhala Day

Sent by Dr.

Material Sputum.

Nature of Examination Culture for A. F. B.

Culture shows no growth of A. F. B.


M. B. S., D. T. M. & H.
CLINICAL PATHOLOGIST F.R.S.T.M. & H. (London)

Dial: { 55-8522
54-4080

Dr. M. C. Paul's Bacteriological Laboratories

131C, BIDHAN SARANI, CALCUTTA-700 004

(Near Shyambazar Tram Depot)

LABORATORY EXAMINATION REPORT

Date of Receipt 8.12.90

Date of Report 8.12.90

Ref. No. 43

Patient's Name Parulbala Dey.

Sent by Dr.

Material Sputum.

Nature of Examination Stained for A.F.B.

A.F.B. ——— Not found.


M. B. S., D. T. M. & H.
CLINICAL PATHOLOGIST F.R.S.T.M. & H. (London)

DR. R. S. BHAKTA

MD. TTD
PRESIDENCY SURGEON

222, S. K. DEV ROAD (CHOUDHURI BAGAN)
PATIPUKUR, CALCUTTA-700 048. TELE : 34-2406
HOURS : 8-8 A. M. DAILY
8-10 P. M. (EXCEPT SATURDAY)

Sant Parul Day

Bronchogenic CA?

Sputum for malign cells / Bronchoscopy

Phenyl 2 sp 3 in 8 mg

Ref to Chittaranjan CA Hosp.

or Trauxapukur CA Hosp

18/1/91

R. R. S. BHAKTA

MD. TTD
PRESIDENCY SURGEON

222, S. K. DEV ROAD (CHOUDHURI BAGAN)
PATIPUKUR, CALCUTTA-700 048. TELE : 34-2406

Hours : 7-8 A. M. Daily
5-6 P. M. & 8-9 P. M. (Except Saturday)

Sant Parul Bala Day

unt x Ray
2 or 3 films
apex.
Cough
Anorexia

Inj Dicainabalin (25) stat.

Ascoril 2 sp 3 in daily

Haematinic one daily

17/4/91

DR. R. S. BHAKTA

MD. TTD

PRESIDENCY SURGEON

222, S. K. DEV ROAD (CHOUDHURI BAGAN)
PATIPURAH, CALCUTTA-700048. TEL: 34-2408
HOURS 10-12 A.M. DAILY
10-12 P.M. (EXCEPT SATURDAY)

vomiting.
Anorexia
anorexia
cough & exp.
febrile episode. 14/11/70
P. 100
140/70
+ dry chest 17/11/70
Stomach congestion.
DLUL
clinical
cramps RU2
Heat & R
Jaundice +
Anemia +
WBC/NP
No enlarged gland

Sent Paralbala my 70 rs

Serum Bilirubin: -1.6

Amphileu (50) 3 hr 2-3 rs

6210 " " " " " " 2 hr 2-3 rs

" " " " " " 2 hr 2-3 rs

my E.T. dinit 2 hr 2-3 rs

my D. cadaveraleu (25) 2 hr 2-3 rs

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" " " " " " 2 hr 2-3 rs

Neotomic Eucema.

4 Mucibail 4 rs at 60 rs

" " " " " " 2 hr 2-3 rs

44/12/90

" " " " " " 2 hr 2-3 rs

" " " " " " 2 hr 2-3 rs

" " " " " " 2 hr 2-3 rs

$\frac{1}{2}$ $\frac{1}{10}$ Sumo x (50) 3 lin daisy x 50 yds
 $\frac{1}{1}$ $\frac{1}{1}$ Siaplex lypin 25, two daisy
 $\frac{1}{1}$ $\frac{1}{1}$ Terpet 3 5, 2 lin daisy
 $\frac{1}{1}$ $\frac{1}{1}$ Mils 2 5, 2 lin daisy
 $\frac{1}{1}$ $\frac{1}{1}$ $\frac{1}{1}$ Ing Doryphylla one ap fair

27/12/90

81/12/90

100
180/75 " "

Cough 1/8

Transit 1/8

Hills 2 sp 3

" " Lar traps 2 sp

1/1000 Placidox-5

1/1000 Adefshane

Sumox (500) 3 hr x 7 2 ss total

Scaflex up 2 sp fair day

Th phyllen (250) one cap fair day

Viscodyne 2 sp 3 hr day

Lar traps 2 sp fair day

Placidox-5 one at 10 hr

Adefshane one alt day after break for 3 hr

Spinal Cell Line
Susceptibility
Preparation

4/11/14
From Culture L.
Susie's 1st
propound

DR. R. S. BHAKTA

MD, TTD
PRESIDENCY SURGEON

222, S. K. DEV ROAD (GHOSHURI BAZAR)
PATIPUKUR, CALCUTTA-700048. TELE: 24-2408
HOURS 6-8 A. M. DAILY
8-10 P. M. (EXCEPT SATURDAY)

Karul bala day

" " Siaplex lypu 20p 3hr deng

— T & phyllis (250) all at 10hr deng

" " " viscopre 20p 2hr deng

— Adaphane all at 10hr deng

100p Placido x 5 all at 10hr deng

" " " Correx 20p 3hr 2p

x Day Chest P.A.

Sputum culture & sensitivity for paratyphoid

In E/dent all at 10hr x 6
Droxy (500) 1hr x 62 hr

21/91

17.11.91

600 Paraxin (500) 3hr x 72 hr

P. 100 600 Microdope all cap 3hr x 62 hr

110/65 88 T & phyllis (250) 1hr 2p

6 210 Metwyle (100) 3hr deng x 2

EOP

" " Siaplex lypu 20p 1hr deng

" " " viscopre 20p 3hr 2p

" " " Correx 20p 3hr 2p

x Day Chest P.A.

17/11/91

Chittaranjan National Cancer Institute
(Hospital)

37, Shyamaprosad Mukherjee Road
Calcutta-700028

PRESCRIPTION

Name... Parnalal Day Tripathi
Regd. No. 2191239 Bed No. 827-D

Date

Adv:
① Chest X-Ray, R & L views.
② Sputum for A-F.B
& malignant cells.
③ Tab Oracip (500mg)
1 tab bdx 7 days.
④ Tab Polysilin
1 tab bdx 7 days.
Total 2 tabs.
2 tabs
23/11/91

Sputum for cytology
& malignant cells.
Tab Baclofen 100mg
1 tab bdx 7 days
x 5 days



47-6033

CHITTARANJAN CANCER HOSPITAL

(A Component of C.N.C.I.)

CALCUTTA-26

Name.....*P. R. Chandra*.....Regn. No.....*121/232*.....Date of 1st attendance.....*12/2/51*.....

Please bring this card with you when attending hospital. If unable to attend on the date stated another appointment should be made. All patients are expected to contribute towards the cost of treatment.

স্বাক্ষরিত **DR. J. BISWAS /**
 হাসপাতালে আসিবার সময় এই কার্ড সঙ্গে আনিতে
 জরুরী নহা। নির্ধারিত দিনে আসিতে না পারিলে অতদিন
 ঠিক করিয়া লইবেন। সকল রোগীকেই চিকিৎসার্থে বরচ
 দিবার অর্থ অগ্ররোধ আনা যাইতেছে।

রোগী ভর্তি হইবার পর পুরা বাড়ীর ঠিকানা এবং
 টেলিফোন, যদি থাকে, নবর লিখাইয়া যাইবেন।

12/2/51

8/2/51 *6.2.51*

6/2 - 8/2/51 *20/2 - 28/2/51*

38188
 ROOM NO. 1
13

14/2/51
For Rm. 1

25.2.51
At 8-30 AM

CHITTARANJAN NATIONAL CANCER INSTITUTE

2/4/8 (HOSPITAL UNIT)

37, S.P. Mukherjee Road, Calcutta -26.

Name Paul ... Bar ...

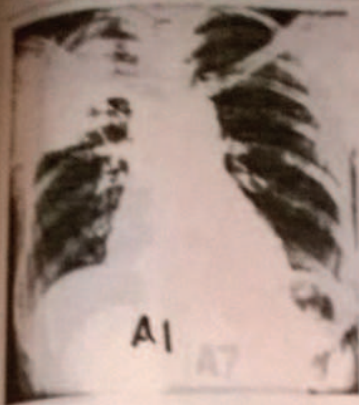
Regd. No. ... S. 91 / 239 ...

is informed to attend the Radiotherapy Department
of the Chittaranjan National Cancer Institute at 10.00
on ... 25/2/91 ... 8-30

MEDICAL SUPERINTENDENT.
(NCI (CENTRAL))

C.O

14/2/91



29. 11. 90



18. 1. 91.



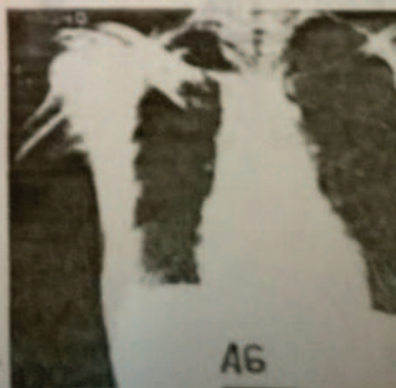
28. 1. 91.



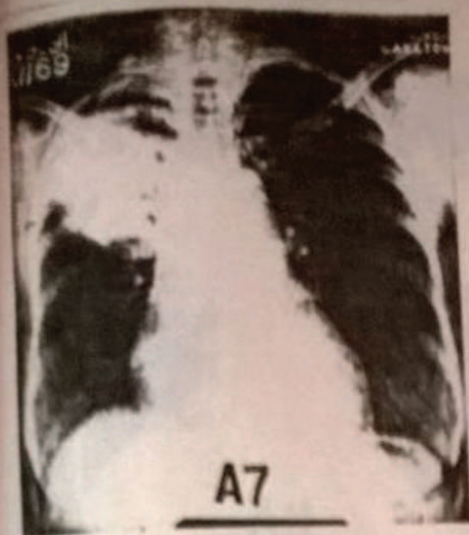
16. 4. 91.



16. 5. 91.



5. 7. 91.



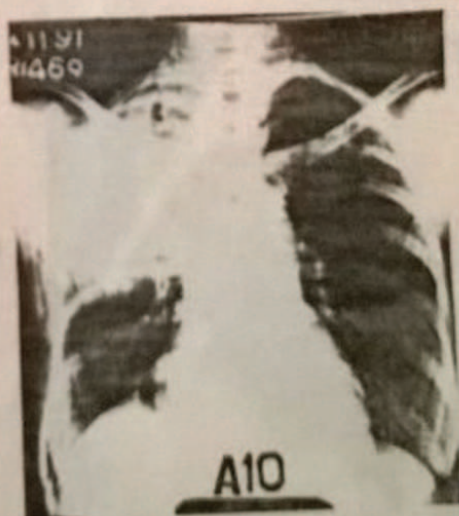
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14. 9. 91.



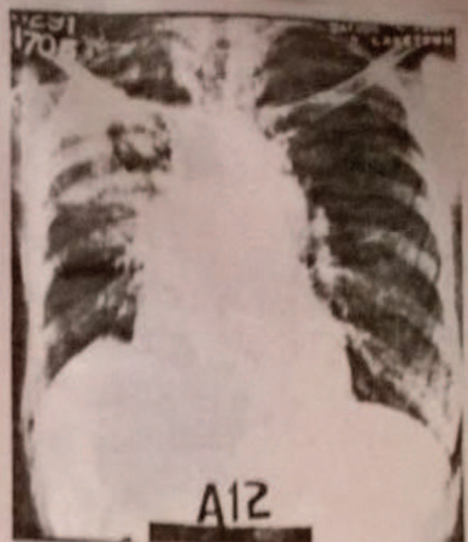
16. 9. 91.



5. 11. 91.



20. 11. 91.



21. 12. 91.



19. 2. 92.



19. 2. 92.



A-15

6. 9. 92.

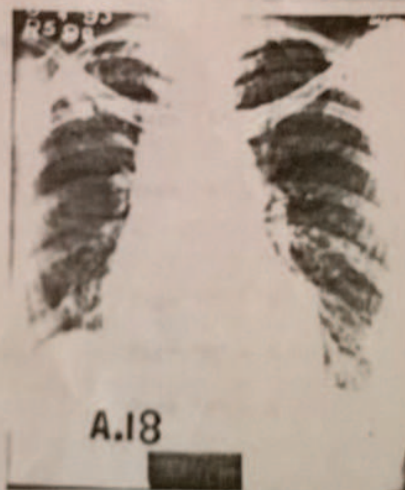


6. 9. 92.



A-17

12. 8. 92.



A.18



A.19





MIDDLE - SMT. PARULBALA DEV
RIGHT - DR. ASIM KUMAR CHATTERJEE

At the same time I was able to completely cure the stomach cancer of Bijoy Kumar Hui. To get his case-papers from Chittaranjan National Cancer Hospital was not at all a problem. The then Deputy-director of Chittaranjan National Cancer Hospital was Dr. Utpala Chatterjee. She helped me in this respect but on the condition of showing my works to her. I did as she said. Then again Dr. Chandra (Onco-surgeon) also helped me a lot. I had cured Smt. Binapani Sarkar, a turned-out patient of Dr. Saroj Gupta, with huge amount of effort, and the details of which I have already provided hereinbefore. Dr. Saroj Gupta was the then Director of Thakurpukur Cancer Hospital. I also had to break through the barriers of Thakurpukur Cancer Hospital and IPGMR Institute. Bijoy Kumar Hui's case has been documented in the paper titled 'A Conventional Homeopathic drug Psorinum has been unconventionally experimented for Carcinoma treatment – experimental outcomes on two patients', which I have incorporated in this book.

Reports and supporting documents of Bijoy Kumar Hui

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CHITRANJAN NATIONAL CANCER HOSPITAL
DEPT FOR OPD PATIENTS

No. 049981 Date of Issue 13/12/53
S.S.K.M. HOSPITAL, CALCUTTA. 4 JAN 1954

Name Bijoy Hui
Age 70y Sex M
Disease 7.

Date	Treatment
4/1/53	Recurrent Vomiting Severe Anorexia w/ w/ h/ t/ Ad Ad DUQI Endoscopy to exclude CA Stomach

Re. 1- (2)

Date	Treatment
13/12/53	553/93 (27/1/93)

Section shows histology of
fascia nodule with proliferation
of glands & vacuolated
epithelial cells in
place. The histology
is suggestive of
malignant glandular adenocarcinoma.

Washed from at 12 noon

(4) DR. ANUP MAJUMDAR

777, 33716, 91 / LUES, THUES, SAT

Re. 1/-

Treatment

TICKET FOR OPD PATIENTS

No. B 0220689

Date of Issue 28 OCT 1997

S S X M

RT/M/13/6, 32

HOSPITAL

Name Bijay Kumar Hui

Age 60 Caste H Sex M

Disease Ca. stomach

Date 28 OCT 1997

Treatment

1/0
unaware
Therapy

1/0

- Endoscopy - 1985
- Ca. stomach (adenocarcinoma)
- 2 biopsies - 1992
- Biopsy showed adenocarcinoma
- Chemotherapy - 1992
- Chemotherapy - 1992
- poor physical condition
- Patient was on chemotherapy
- since 1992

Endoscopy + biopsy 1/3/93/95
5/4/97/93 → No metastasis
physically fit

HE

G.C. below average
An + cy + ci + o
No supraclavicular lymphadenopathy

(2)

(3)

Date	Treatment	Date	Treatment
	<p>Abd. no abs. lump palpable.</p> <p>lower just palpable.</p> <p>Abd.</p> <p>Blood. TC cc. Wb/ ESR.</p> <p>LFT</p> <p>uric creatinine</p> <p>stool. OPC occult blood</p> <p>USA. upper abdomen</p> <p>chest x-ray. PA.</p> <p>Reviewed ch. <i>frump m. 1/2</i></p> <p>stop ED to <i>continue</i></p>		
	<p>To attend Eye OPD</p> <p>for opinion regarding eye ailments.</p> <p><i>frump m. 1/2</i></p>		

Here I would like to mention that the amount of support shown towards me by Shri Shankar Sen, then MLA and Minister of Science & Technology, then Secretary, Prof. Dilip Bose, the Dean of Medical Faculty of Calcutta University and renowned pathologist, Prof. Subir Dutta, and Dr. Amiyo Kumar Hati was beyond any description. I can humbly bow my head with great respect for all of them. On their encouragement in 1995 in the Second West Bengal State Science Congress on 28th February – 2nd March, 1995, Kolkata, case study of two of my patients was published. We had sent almost 50 documented case studies out of which two were selected for publication by the committee. Among those 50 cases, in some patients cancer had mostly regressed, while some were under treatment. I will always remain grateful to Dr. Ashokananda Konar and Dr. Subrata Bhattacharya for two reasons. They both are very honest and dedicated practitioners who for the greater good accepted my point at the very first time and acknowledged the fact black and white where it was clearly mentioned that said S. P. Deb Roy had cancer and it had regressed under my treatment. Secondly, both of them are absolutely fearless persons. As they had given me an acknowledgement, they had to face the consequences as well, but they clearly declared that they had seen the cancer patient and the patient had been cured by me.

Many of our papers have been published so far. I would like to attach two of those papers in this book in order to make things more clear to the readers. Whenever I imagine the amount of effort I had to put in writing these papers and to spend a huge sum of money in collecting documentary evidences, I remain astonished. I had to convince several personalities of the scientific community. In one of the papers I have tried to bring about all the issues related to cancer, and in the other I have modelled the treatment method in a new light. One may have to deeply analyze all the scenarios in order to understand the subject.

Science is gradually moving ahead with time with new researches, developments, discoveries, inventions and innovations. In last 35 years I have come across a huge change in the conventional treatment methods, in the rules governing the scientific community and in the individual approach in treating a particular disease. I wish to see better results in future as well. It is very important for all of us to adapt ourselves and also create a module which would adapt and adjust itself to the present as well as the future cancer treatment models.

I have already reached only one end, i.e. the total regression. I want the entire world to see it and believe in my treatment model. There lie still for me miles to go to reach a comprehensive model consisting of preventive measures to pre-empt the occurrence or invasion or attack of such disease, measures for non-occurrence and a module of healthy habits or practises to keep away such silent killer of the humanity.

The nucleus I have created, its creation and development are based on certain principles and ideals. It has not come through any confrontation, but have generated out of mutual respect, faith in each other, belief and a unanimous vision to serve humanity and the entire mankind.

Problems Of Rehabilitation

Rehabilitation is a pertinent issue in the realm of Cancer –

Of course we can neither behave like a sky-diving bird nor like an ostrich which pushes down its head whenever it senses any threat. The word 'Rehabilitation' has a social and religious sanctity and is associated with a numerous chain of events and activities resulting from any eventuality. It is said that 'Cancer has its beginning but no end'. This means that even after the death of a cancer patient, its remnants are no less pernicious. Not only the human asset is destroyed, but the financial solvency as well as the monetary resources are fully jeopardized, making the life and living of the future generation miserable. Proper rehabilitation of the family of the deceased needs to be oriented very carefully.

In case of demise of a propertied person, the problem of rehabilitation is no less important. It is rather different depending upon the occupation of the deceased. A factory owner's family has to see how the factory will again be set in motion, keeping all commitments towards employees and others intact. The family of a businessman will have to collect back unrealized dues, if any and to renew contracts with new clients honourably. Sometimes in the case of death of a highly paid salaried person in a private firm, the task of rehabilitation points to the realization of the due amount payable to the deceased's family and to take care of the amenities available to his/her dependants, as well as to any best investment of funds for family's benefit possible. Actually the word rehabilitation includes all the above activities to be performed with full responsibility and with a practical and humanitarian outlook.

Rehabilitation and Counselling go hand in hand –

Since cancer-affected families suffer from a traumatic experience from the very moment of outbreak of the disease, entire mental balance of the family gets disturbed, and care should be taken to protect the female members from all kinds of harassments inside as well as outside the family and to save them from any financial loss also. On the other hand, rehabilitation of the poorer section of the people needs gainful employment. Rehabilitation activities either by consultation or by financial help should always be based on reality.

Rehabilitation starts from the very moment of detection of the disease in a member in the family. To avail of the pathological tests and treatment of the disease in the urban centre, temporary residential arrangements for the patients along with attendants are one of the very demanding requirements. Other important measures are enlisted here-in-below –

1. Formation of Official Rehabilitation Committee in the Government level on behalf of Social Welfare Department or Municipality or Anchal Panchayat is solicited to propagate and accelerate the programme.
2. Arrangements for Terminal Centre are necessary where there should be provision for attendants to stay with the patients.
3. During treatment, minimum financial support will be required in some deserving cases.
4. Counselling will be necessary for systematic management of a patient's financial resources during treatment.

5. To create awareness amongst the family members about the situation that may arise after the death of the patient.
6. Suggestion about the management of the property of the patient, if any, in the event of his/her death.
7. To take some emergency measures like job-oriented training of capable members of the family depending on their suitability.
8. Besides monetary problems, man-management and education of the coming generation should also get its proper priority.
9. To assist the patient's family to meet urgent family-commitments as well as socio-religious commitments with a view to give mental boost to the patient, which may help in his/her possible recovery.

While we are going to address the entire problem in a comprehensive manner we should not forget that prolonged colonial exploitation of our people has ceaselessly tried to rob off our dynamic attitude, originality of thought and indomitable courage for taking new initiatives and ventures. We fully understand that unless a cost-effective method to fight against Cancer is discovered, we are sure to ruin. So why should we not try with new ventures like Psorinum Therapy which can open a new vista with infinite potentiality?

To achieve this target we shall have to give a dip into the deep ocean of our rich heritage and procure precious stones and pearls carefully nurtured so long in the age-old tradition of our country. It has been proven historically that people of our country never shake off their social responsibility whenever any situation demands.

Right from the days of Maha Prabhu Sri Chaitanyadev, the great souls of Raja Rammohan Roy, Ishwar Chandra Vidyasagar, Kaviguru Rabindranath Tagore, Swami Vivekananda and others in different ages and in different fronts appeared in national scene and propagated the great principle of sharing the social burden with others. The glittering history of national freedom struggle embedded with the scintillating instances of self-sacrifice and martyrdom corroborates the same thing. In the days of partition of India what a glorious role was played by the innumerable families of West Bengal to accommodate the refugees and share the liabilities of their friends and relatives who migrated to India helplessly, as if the head of the family is providing shelter to his near and dear ones in their peril as far as practicable and trying to give them minimum possible support for their survival. Definitely there are other exemplary instances in so many parts of the country too.

In the light of a pragmatic approach real remedy can be achieved though the network of related health centres. By virtue of this method of cost-effective treatment through Psorinum Therapy, the sharing of responsibility from all concern including public enterprise, private enterprise and philanthropic institutions, India can take a lead in the treatment of cancer both in theory and in practice.

In recent days we have also been experiencing a peculiar phenomenon that in most of these cases relatives of the cancer-patients chose to conceal the name of this dreadful disease from the victims, apprehending the extent of the latter's frustration and mental agony. Entire rationale behind this phenomenon is to give mental peace to the patient and to bear the huge

psychological and financial burden of the disease in a collective way, which reminds us the legacy of sharing risk in our joint family system. This type of practice is in reality prevalent more in the tribal states of the Northeastern part of India.

Though at present most of the families are of nuclear type, settled at different places, still that collective spirit and approach to the calamity or family-disaster of course work uniquely if they with their limited resources try their best to extend their hands of cooperation. This kind of help will be more forthcoming if the doctor and his team give them proper counselling and describe how economically and with austerity measures the Psorinum Therapy is designed for the treatment.

So in this area of counselling we have to break the shackles of mechanical adaptation of western model of typical doctor-patient relationship. On the contrary we are to imbibe the noble and collective spirit of humanism implanted in our glorious Indian culture and skilfully blend the advanced research and technological contributions of the developed western countries.

The balanced and realistic approach will pervade everywhere cutting across the narrow boundaries of different nations and countries and will successfully pass the acid test of time being accepted perennially at the international level.



Khoma Das and her daughter with Dr. Asim Chatterjee



A rehabilitated patient with Dr. Asim Chatterjee

Need For A Terminal Centre

After overcoming many difficult times and crossing the barriers of religion, caste and socio-economic structure, I have gained the experience of administering an organisation diverse in nature. I had a short experience of handling the anti-social elements of the locality and involving them in the colossal task that we had in hand. This experience came handy when I worked towards formation of a few NGOs one after the other. The zigzag way through which I had to walk, these NGOs played a very important role. Firstly we created the 'New Resource', then 'Oncolink', followed by 'New Horizon Centre for Cancer Research and Treatment', 'Combat Cancer' and later on 'Rabindranath Chatterjee Memorial Cancer Trust'. Apart from these I also worked towards the sustainable progress of 'Subodh Mitra Cancer Hospital' and 'Gandhi Seva Sangha'. NGO defines an idea and a handful of above-self people dedicate themselves to bring that idea to reality. The process of administration of any such NGO, social and voluntary, is very tedious and demands for huge amount of time as well as unending and firm determination. These NGOs and related infrastructures are created to combat certain critical situations, to take the enormous task forward, and to straighten those zigzag ways for the future generation to embark upon a comparatively easy journey.

Many issues are involved in the establishment of a terminal centre of cancer treatment. In the developing countries the treatment of upper class, upper middle-class and lower middle-class people are performed under the same roof. But things start getting more challenging when the treatment of poor people is thought of at the same facility. The poor people earn marginal wage mostly at a subsistence level and their spending capacity is very limited, next to nil. In our country most of the problem arise in such cases, because 20% of the population, although they stay in this country, are hardly recognised as the actual wealth of our country. In our country we believe in serving our guests with whatever all we have, ourselves even staying without food. Women in our house gain pleasure draining out all their energy by working for long hours. Their working hours start at 5.00 o'clock in the morning and end at 11.00 or 12.00 o'clock at night. They also bear the main responsibility for the well-being of the family. This includes providing water and firewood, often carried from long distances, used for drinking, cooking, cleaning and washing. Women also bring up the children and take care of aged members of the family. They also help at weddings and funerals. Usually women grow the food needed by their families, through hard labour in fields which are often situated far away from their villages. Women in urban areas also often work long hours for low salaries in factories, offices and domestic service sector far away from their homes. And all these responsibilities she bears upon her shoulders without any complain. Because she loves her family and there is a hunger of respect, that is the only thing she expects in return. She is the person who belongs to this country, knows where this country's real wealth is, our love and respect, our culture, our patience, our dignity, our forgiving nature, our pride, all these are a part of all our lives.

I have always said that 60% of the cancer patients in India do not depend on the government aid for their treatment. They fight with this deadly disease staying in their

community, in the midst of well wishers, and will continue to do so. Our work would be to analyse them and empower them more, so that they can face the situation more effectively. I have come to this realisation after observing our social and economic scenario for so many years fighting against cancer and looking for cancer patients, by frequenting to the remotest corner possible.

Let me give a small example (not related to cancer) to demonstrate self-sufficiency of a poor household: Born in a poor family in a slum in Kolkata, Mamuni Biswas' father was a rickshaw puller. Her younger brother and she were very young when their father passed away. Mamuni Biswas after a lot of struggle completed her class -X examination, and then she took training for nursing and now she has been working at CCMRCC for more than eight years. Now she is 28 years old. On the other hand, her brother, Shankar Biswas started pulling rickshaw at a very tender age and left his studies. First when I met him he was complaining about his elder sister as being very literate and in disagreement with him most of the time. He also said that he used to earn around Rs. 500 – Rs. 700 per day. When he was only 19 year old, he got married although he faced refusals from his family. Now he stayed away from his parents but with his wife. He said that his elder sister would be now 28 year old and unmarried because she was educated and working. She was asked to get married when she was 16 year old but she did not. Now that she has become self-dependent she would like to stop his brother's wedding. I asked him what would happen if he ever fell sick. He replied that his wife working as a domestic help would not have any problem so far.

In our country, almost 70% of the people who cannot gain higher education, people who stay in villages and who are self-sufficient, silently do their work, but when literate people do not get proper job, there rises the problem of unemployment. The cancer patients who come from the economically and educationally poor background who lived on their own throughout their lives, receive tremendous support from their community where they are properly looked after. Thus we have to build our infrastructure in this backdrop.

Well-to-do people come to us after receiving unsatisfactory or failed treatment resulting from the conventional method. We help the patients to overcome the bad condition they were into as quick as is reasonably possible. Patients who are expected to live for hardly seven days or so are taken care of to get extended their life up to 6 months or even up to 1 year. It is hard to ignore the conventional treatment method in the prevailing social, economic and financial scenario. Conventional treatment has a pre-estimated budget and stands on an expectation of possible future death with very low probability of recovery. Patients during the last days like their family-members, wish for a peaceful and painless exit if possible. And if the patient survives, it is a miraculous achievement. Today when people are so busy in their life, it becomes a problem for them to spend long time for such treatment in cases of aged as well as young person. We try to create a treatment model keeping their respective needs in mind. Every individual requires due care and attention, while at times it becomes very difficult to keep such patient at home. The family members cannot abandon him as well. In this situation they express their anguish towards us. There are a few examples of such behavioural pattern I have come across in my journey.

On Our Social Responsibility

Social Responsibility to Treat Cancer in the Third World' –

Cancer only reminds of death. When the biopsy report is positive, the family members try hard to hide the news from the patient. In case of an elderly person, although it is comparatively easy to slowly accept the news, still it is very difficult to continue the prolonged process of treatment after knowing the ultimate fate.

It is next to impossible in the case of a younger person. Attempt is there to save the life even at the cost of the last penny. Most important thing in this situation is to properly deal with all the problems regarding the disease and the effect of medicines and above all to pursue a responsible process of treatment without any haphazardness.

Lack of mental and financial strength of the family of the patient moving gradually towards the jaws of death due to a disease with no full-proof medicine or proper treatment, takes us to a very disastrous situation. The ongoing effort across the world to invent a Cancer medicine however gives a ray of hope. But until such a medicine is invented, it is the duty of the doctors to think of a way out for temporary relief of the patient as well as the family members. In many cases, the patient-family should be given detailed information of the conventional ways to treat Cancer i.e. Surgery, Radiation Therapy or Chemotherapy and about the fatal stage of the disease so that the family can take practical decisions depending on their financial strength or man power. No doubt, doctors are responsible to fight for the patient's life till the last moment. So it is hard to keep a balance between the financial capacity of the patient's family and the logical decisions of a doctor in the process of treatment. All aspects of third world penury should be closely taken into consideration while one is associated with cancer treatment vis-a-vis performing the duty of social responsibility.

A doctor has to keep in mind the financial capacity of the patient's family in a country which fails to supply even safe and clean water to a patient, keeps electrolyte balance with salt-sugar water, and often accommodates 500 patients in a hospital with an actual capacity of 100. The fact remains that having been aware of the absence of any medicine for Cancer, only a small proportion of doctors have devoted themselves to the treatment of the disease. None of the methods to treat the disease till date claims an absolutely complete cure. Besides the conventional treatments, arrangements for ultra-sonography, endoscopy, different tests of blood and body-parts together with the supply of liquid food, blood, glucose, and saline are inevitably necessary.

People are aware that without the above-mentioned arrangements, no patient can be treated properly. Priority demands to arrange for these things rather than buying costly Chemotherapy doses. Quite often lack of financial strength causes rapid death of a cancer patient. This proves that a practical, sensitive, sympathetic advice of a doctor is much more useful than a costly medicine. Any indifference and apathy on the part of the doctors may give birth to anxiety and uncertainty among the general mass, regarding medical practitioners.

An incident can be cited here where one Lecturer aged above 30 yrs. of a college in Kolkata did hide his positive biopsy report from his family as he was aware that his family was financially too weak to get his Cancer treated. So he preferred death on his own. There are hundreds of such instances where an educated and even high-income-level cancer patient prefers economic stability of his or her family left undisturbed to his prolonged treatment for uncertain recovery.

Everybody is aware of the fact about Cancer that it is very much different from other diseases. Sometimes it may be cured having been detected at an initial stage, but sometimes all conventional and non-conventional processes of treatment may go in vain. There is a common belief that cancer may be cured if detected at an initial stage, but it is not always true. The disease at regions like pancreas causes death even if it is detected early and all scientific methods of treatment are applied in time.

So we need to know the life-span of a patient in each depending on part or organ of the body affected by the disease. We need to be aware of all effects of cancer on different parts of the body. Most dangerous parts of the body, if affected, include liver, pancreas, gall bladder, stomach, oesophagus and lung. Other less harmful places are kidney, prostate, uterus, breast, ovary, bladder, testis etc. Life-span of a patient is much more in the second type of cases than in the first ones.

However, Radiation Therapy sometimes comes as a government aid and it is less costly also. But Chemotherapy drugs are available at both higher and lower prices from external sources. The so-called media-therapy i.e. advertisements to cure Cancer, occasionally gives a misdirection and comes with a false hope of survival through a short-cut method.

In the context of all the above-mentioned social conditions, I appeal to the doctors to do away with the misconception and wrong information about Cancer, which regularly leads to confusions in treatment process. Besides a group of efficient and devoted doctors, a group of corrupt people often try to destroy the entire medical system in naked greed for money and immediate gain in business.

In this complex situation some medical scientists have undoubtedly been rendering yeoman service by devoting their time and energy in the process of inventing Cancer medicine, while others, mainly social activists concentrate their attention on health, finance and other necessities of supplying food, blood, glucose and saline to the patient, in order to cure such patients, and in very rare cases, they both combine in one entity.

The last factor to be remembered is that where a cancer patient is bestowed with a family, it is absolutely necessary to think practically before spending everything for a person who is destined to die in near future in all probability.

Cancer in a Graceful Eye – A Harsh Reality'

Cancer is a dangerous disease which, since ages, has affected India and the rest of the world in the form of an incurable disease. There has been an ongoing process of research on it in different parts of the world. But practically there has been no big success. The most

1. This is the translation of Dr. Asim Kumar Chatterjee's article published in a Bengali magazine named 'Srayan'

important point is that this disease does not involve any bacteria or virus. Diseases like Aids, Typhoid, Cholera, yellowfever, T.B. – all are caused by either bacteria or virus. Cancer can be first detected in any part of the body and gradually it spreads from that portion of the body to other. Let us take the example of liver. This is an important body part. It has many significant functions to perform. What happens when it is affected by cancer? It no longer stays as the normal liver, but turns into a hard tumour. Nobody except God can bring back the liver. Moreover, the disease would not be confined only to liver, but may spread to other parts of the body. This is called Metastasis. Another point to be noted is the above-mentioned diseases like AIDS, TB, Typhoid – all are either of 1 or 2 types. But cancer is of as many as types. 120 types, although physicians do not classify it into so many types. Scientists have indeed classified cancer into many more types. Those are much complex issues but we would not enter into those complications.

Conventionally there are 3 ways to treat cancer – surgery, chemotherapy and radiotherapy. But if the problem is in some critical parts of the body, surgery is not possible. Other types are at times RT and CT resistant. In these cases, patients have no other way but to die under the conventional treatment procedure.

Treatment of cancer is highly technical and very expensive. This is because, the recognised ways of treating cancer are all invented in the developed countries, where the financial problem is not a big issue. Unfortunately the persons who can bear the cost are only 5% in India. Another 5% may afford the treatment at the cost of their other necessities. But in general, people of third world countries where per capita income is very low, cannot at all afford the treatment. Generally those with monthly income less than Rs. 25000-30000 per month face huge difficulties to handle the disease. About 90% of the Indian population is unable to continue cancer treatment. It is impossible to balance the cost required under the present medical system with cancer treatment.

A saying is there that if a country's problem is not dealt by its own inhabitant on its soil and with its own resource, then the country's problem cannot be solved. Unfortunately none of the researches whether govt. aided or private is taking initiative of treating the economically backward section of the Indian population. Everybody is hyper about cancer; everybody is hyper to do something. But nobody knows what to do. This is not a toy. Nobody is aware of the actual problem. But the scientists have provided warning that by 2020, cancer will be an epidemic in India and China. The very hard truth is, there has been no government or private Initiative in India to provide minimum treatment to the 90% of the affected population. And 7% out of 10% of the remaining affected population will be totally ruined under the influence of highly technical treatment procedure. The harsh truth is that they would be served by their beloved ones to the best of their abilities.

DR. ARADEEP CHATTERJEE

Dr. Aradeep Chatterjee, my son, from his early childhood days took deep interest on the subject of cancer. By the 18th year of his life he already gathered the basic knowledge of

1. English Version of the article in Bengali published in the magazine, Atmabikash, 1st Issue, October, 2005.

cancer and its treatment. He has played a significant role towards the development, clinical trials and dispatch of the drug 'Psorinum Therapy' to the cancer patients. Launching of this unconventional drug on various international platforms is a result of his untiring efforts, and his contribution in this field calls for applause. Aradeep had sent the case studies to NCI (National Cancer Institute) which is under the National Institutes of Health in the US. The then Director of NCI, Jeffrey D. White, MD, also visited us in India, here at our facility viz. Critical Cancer Management Research Centre and Clinic (CCMRCC), and invited Dr. Aradeep to present a paper on the subject with reference to the Psorinum Therapy. Dr. White was amazed at Dr. Aradeep's understanding of the subject. He was acknowledged to be the first medical student from India to have successfully performed the Best Case Studies (BCS) programme in USA. He was also invited to join the Advisory Panel Review of Cancer Therapy Evaluation programme of the Drug Development Group held on 31st October, 2005 in Maryland. Later on, the American Society of Clinical Oncology (ASCO) selected the research details of Aradeep and his colleagues and presented it at the 2009-ASCO Annual Conference held on 29th May – 2nd June, 2009 in Orlando, Florida, USA. Aradeep was the lead study author and at the conference he stated, "These types of cancer are called silent killers, usually diagnosed at the end stages. The bad news is that till now, conventional cancer treatments can rarely cure a patient having these types of cancer. Now, the good news is our research shows Psorinum Therapy is effective in treating stomach, gall bladder, pancreatic and liver cancers. Complete & partial tumour response to Psorinum Therapy as well as overall 5 year survival were observed in a significant number of participants indicating that Psorinum Therapy can change or substantially improve the oncology practice in near future. The research study also confirms that Psorinum Therapy is non-toxic, which would be a great relief to the cancer patients." Aradeep has also gained the membership of ASCO. He is associated with many national and international publications till date.

Dr. Aradeep has played a great role in writing the book 'The Management of Cancer in Totality – India can take a lead' especially by writing the core cancer-related portions of this book. He also has put in many valuable ideas on the subject and also on how the book should be designed. Many patients who have been treated at CCMRCC have been cured following his treatment module. Presently he is working in tandem with the Chemo-Prevention Department of Chittaranjan Nation Cancer Institute (CNCI) along with Prof. Jaydeep Biswas and Dr. Sudeen Bhattacharya. In the meantime, three important papers have been published under this unique joint-initiative. At this point of time, Dr. Aradeep Chattarjee is the Director of the CCMRCC. Since this book describes my 36 years long journey from 1980's up till 2005, there were many political as well as social challenges that I had to face. Dr. Aradeep was never a part of this turmoil. Hence, I do not feel his name should be associated with this volume of the book as a co-author. I here propose that the following volumes of this book would be published under his 1st authorship and I would step down and would be fairly visible as the 2nd author.

I look forward to highlight more on Dr. Aradeep's works and contribution in this field in the forthcoming publication of ours if any.

American Society of Clinical Oncology

*To all whom these presents shall come, Greetings
Be it Known That*

*Aradeep Chatterjee, MD
having demonstrated an especial interest in the diagnosis,
treatment and care of the patient with cancer is hereby
elected a(n)*

*Full Member
of this Society*

October 01, 2009

Clifford Hudis MD

President

ASCO
American Society of Clinical Oncology

Don C. Cella

Treasurer



CHITTARANJAN NATIONAL CANCER INSTITUTE

Golden Jubilee Celebration

1957 - 2007

**SYMPOSIUM ON
RECENT TRENDS IN CANCER RESEARCH & TREATMENT**

PROGRAMME

Thursday, November 01, 2007

Mini Hall

8:00 – 12:30 REGISTRATION

9:00 – 10:00 Inauguration

10:30 – 11:15 Oration P B Desai

11:15 – 11:45 Indraneel Mitra

11:45 – 12:15 H S Shukla Cancer control and prevention

12:15 – 12:45 Rajesh Alwat Endosurgery & robotics in prostate cancer

12:45 – 13:05 P Jagannath

13:05 – 13:25 Kalyan Sarcar Breast cancer

13:30 – 14:00 Mid Break and Discussion

14:00 – 16:30 Session I: Molecular Oncology

14:00 – 14:30 **Subodh Mitra Oration** M Siddiqi

14:30 – 15:00 T Rajkumar

15:00 – 15:30 B C Das

15:30 – 16:00 B R Das
Evolving technologies: ushering a new era for cancer diagnostics
A novel role of bHSH transcription factor activator protein-2 alpha (AP-2 α) in cell cycle regulation mediated control of myogenesis and rhabdosarcoma formation

16:00 – 16:30 K Somasundaram
Therapeutic significance of osteopontin, a member of sibling

16:30 – 17:00 G C Kundu

ubilee Celebration

Friday, November 02, 2007

15 Minutes Oral Presentation of Senior Students/Post-Doctorals

		Hall C
9:00 – 11:30	Session I: Cell Signalling & Molecular Mechanism of cancer	
10:00 – 10:15	Dona Sinha	
10:15 – 10:30	Sutapa Mukherjee	
10:30 – 10:45	Aniruddha Banerjee	
10:45 – 11:00	Ruma Dey Ghosh	
11:00 – 11:15	Nabendu Murmu	
11:15 – 11:30	Illa Das	
11:30 – 11:45	Goutam Chakraborty	
11:45 – 12:00	Aradeep Chatterjee	

Aradeep Chatterjee

From: "The Lancet Asia Medical Forum" <TheLancetAsiaMedicalForum@xmr3.com>
To: <aradeep_1@vsnl.net>
Sent: 04/16/2007 01:34 PM
Subject: Less than 1 week to The Lancet Asia Medical Forum 2007. Register Now!



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You have **heard** about us...

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Representatives from the following organisations have registered...

- Cancer Center of Southeastern Ontario, Canada
- Cancer Research Initiatives Foundation (CARIF), Malaysia
- Chonburi Cancer Control Center, Thailand
- Department of Clinical Oncology, Sri Lanka
- Hong Kong Hospital Authority, Hong Kong
- Indira Gandhi Memorial Hospital, Maldives
- Institute of Hematology, Israel
- International Islamic University of Malaysia Breast
- National Institute of Cancer, Sri Lanka
- NCI Cancer Hospital, Malaysia
- Peter MacCallum Cancer Centre, Australia
- Prince of Wales Hospital / CUHK, Hong Kong
- Queen Mary's University, UK
- Rajiv Gandhi Cancer Institute, India
- Royal Prince Alfred Hospital, Australia
- Sapporo Medical University, Japan

04/17/2007

Centre, Malaysia

- Johor Specialist Hospital, Malaysia
- Juan Canalejo University Hospital Trust, Spain
- Koo Foundation Sun Yat-Sen Cancer Center, Taiwan
- Korea Research Institute of Bioscience & Biotechnology, South Korea
- Lopburi Cancer Control Center, Thailand
- Ministry of Health, Brunei
- National Cancer Centre, Singapore
- Sen-en General Hospital, Japan
- Shaukat Khanum Memorial Cancer Hospital, Pakistan
- Subodh Mitra Cancer Hospital, India
- Sydney Radiotherapy & Oncology Centre, Australia
- Taipei Medical University, Taiwan
- The First Hospital Affiliated to Tianjin University of Traditional Chinese Medicine, China
- The West Clinic Excellence Cancer Center, Singapore

... and many more

Certificate of Participation

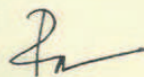
This is to certify that

Dr Aradeep Chatterjee

has participated as a delegate at

**ASIA MEDICAL FORUM
THE LANCET 2007**

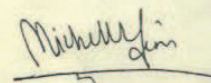
21 - 22 April 2007 • SUNTEC Singapore



Dr Richard Horton
Editor
The Lancet



Lex Rozenbroek
Managing Director
Elsevier Health Sciences Asia Pacific




Michelle Lim
General Manager, Singapore-Malaysia
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THE LANCET Oncology



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CNCI

Chittaranjan National Cancer Institute

37, S.P. Mukherjee Road, Kolkata - 700026

Phone : 2475-9313; 2476-5101(Extn.309)

Fax : 2475-7606; email cncinst@vsnl.com

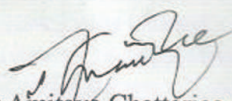
To
Dr Ashim Chatterjee
381, S. K. Deb Road
Kolkata-700048

Date: 07.05.2007

Dear Dr Chatterjee,

Our Institute has a tradition inviting Doctors and Scientists those who are actively engaged in basic and clinical research with the disease cancer. Its my pleasure to invite you to give a seminar at our Institute on May 10, 2007 at 3.00 PM on your research findings in the treatment of cancer patients. Please confirm the title of your talk.

Yours Sincerely



Dr Amitava Chatterjee

Officer-In-Charge (R)

PSORINUM THERAPY IN TREATING PATIENTS WITH ADVANCED NON-SMALL CELL LUNG CARCINOMA (NSCLC): A PHASE-II SINGLE ARM CLINICAL TRIAL

A. Chatterjee Critical Cancer Management Research Centre and Clinic,
Calcutta, India,

J. Biswas Chittaranjan National Cancer Institute, Calcutta, India,

A. Chatterjee Critical Cancer Management Research Centre and Clinic,
Calcutta, India,

S. Bhattacharya Chittaranjan National Cancer Institute, Calcutta, India,

A. Chakraborty R G Kar Medical College, Calcutta, India.

R. Mukherjee Critical Cancer Management Research Centre and Clinic,
Calcutta, India,

R. Bhakta S. Das Critical Cancer Management Research Centre and Clinic,
Calcutta, India,

Purpose/Objective(s): We prospectively studied the clinical efficacy of an alternative cancer treatment “psorinum therapy” in patients with advanced non-small cell lung carcinoma (NSLC).

Materials/ Methods : Our study was phase II, open-level, single arm, and single stage. Participants’ eligibility criteria included (1) histopathological or cytopathological confirmation of NSLC; (2) inoperable; (3) no prior chemotherapy or radiotherapy; (4) Karnofsky performance status between 40 and 70. The primary outcome measures of the study were (1) to assess the radiological tumor, response rates (using CT scan procedure and following the RECIST criteria); (2) to assess how many participants survived at least 1yr, 2 yrs, 3 yrs, 4 yrs, and finally, after 5 yrs of the study. The secondary outcome measure was to assess the side effects of the investigational anticancer drug (Psorinum) if any. The drug psorinum (an alcoholic extract of scabies, slough, and pus cells) was administered orally at 0.02ml/Kg body weight/day as a single dose in empty stomach and ongoing for complete course duration of 2 years to all the participants along with allopathic and homeopathic supportive cares.

Results : 95 participants included in the final analysis at the end of the study. According to the AJCC TNM staging system, 58 (61.05%) of them diagnosed at stage IV. According to the RECIST criteria, complete tumor response occurred in 19 (20%) cases and partial tumor response occurred in 28 (29.47%) cases, 82 (86.32%) of them survived at least 1 yr, 70 (73.68%) survived at least 2 yrs, 58 (61.05%) survived at least 3 yrs, 49 (51.58%) survived at least 4 yrs, and 42 (44.21%) of them survived at least 5 yrs. These participants did not receive chemotherapy, radiation therapy, or any other investigational cancer treatments. Participants reported no side effects from the drug psorinum.

Conclusions : The results of the study show clinical efficacy of psorinum therapy in

treating patients with advanced NSLC. The investigational drug psorinum does not have any adverse side effects. Randomized double-blinded clinical trial should be conducted for further investigation of this alternative cancer therapy in treating NSLC.

Author Disclosure Block : A. Chatterjee : None. J. Biswas: None. A. Chatterjee: None. S. Bhattacharya: None. A. Chakraborty: None. R. Mukherjee: None. R. Bhakta: None. S. Das: None.

**This article earlier published in Journal of Thoracic Oncology, Vol. 5, 2010, Article no. 161,*

Release Of The First Book At Book Fair, 2014

Every now and then my well-wishers used to ask me to pen down all the experiences in my journey and to bring to light a few of the proven cases that are worth mentioning and have contributed significantly towards my search for a cure from cancer. There is a lot of difference between writing a research paper and a book. The person who first encouraged me to do so is Mr. Sriman Chakraborty. I got to know him since the time his father was admitted here as a cancer patient. We did our best to save him; however, as fate could have it, he lost the battle. After some days we came to know that his mother also fell sick and later was diagnosed with cancer. She was also admitted here and eventually her cancer was got regressed. When Sriman came up with the suggestion of writing a book I told him that I would not be able to take out so much of time. A very renowned literary person, Shri Debesh Roy also encouraged me. Repeated pursuance was coming from Prof. Subir Dutta as well. Finally I agreed. Sriman used to come at my house regularly, take down notes and used to work on them nights after nights. Since he was a student of History, at the beginning it used to be difficult for him to understand the subject but with time he got to the insight of the situation. Earlier Shri Pushpendra Bhowmik used to help me write a few nationally and internationally recognised scientific papers. He and Prof. R. S. Bhakta had once accompanied me to Delhi regarding the presentation of a paper. He also played a crucial role in the writing and publication of the earlier book. Under the initiative of Shri Debesh Roy and Shri Pushpendra Bhowmik I began my work. During that time I received constant support from Prof. Dipankar Dasgupta, Prof. Jaydeep Biswas, Prof. Anup Majumdar and many others. I give the entire credit of publication of the first book to Shri Pushpendra Bhowmik. He has always been acting as a pillar of support for me.

With Shri Bhowmik's constant efforts my first book was launched at the 'Book – Fair 2014'. In that event many renowned people made their presence felt like Prof. Jaydeep Biswas, Director of Chittaranjan National Cancer Institute (CNCI), Prof. Subir Dutta, Mr. Prabir Mukherjee, Dr. Subhash Ghosal, renowned anthropologist, and Mr. P. Chakraborty, ex-Principal Secretary, Mizoram Government. The event was also witnessed by Mr. Gautam Saha who always stood by my side, Mr. Nilanjan Bhowmik who silently worked for me and in the past, had always been there at my rescue. Mr. Tapan Sen, Dr. Debabrata Malakar and Dr. Hiranmoy Mukherjee also graced the occasion with their kind presence. The kind of

support and encouragement I have received from my family cannot be expressed in words. My daughter-in-law, Swarnali Chatterjee, and my son, Aradeep Chatterjee, played a very important role in this regard. On that day the opening song was presented by my wife, Mrs. Ranjana Chatterjee. My wife stood by my side at every moment, Swarnali's younger sister, Dew, also deserved praises for all her efforts put in it. It was a very emotional day for all of us. Some of us had tears in our eyes.

One name I would specially like to mention here is Mr. Pradip alias Khokan Chatterjee. He was at that time suffering from cancer and was admitted at our facility. He was so sick that he could not even move on his own. He was a publisher and the book's ISBN no. was arranged by him. Without him it would have taken a lot more time to publish the book. Before the publication I did not have any knowledge about the publication side. The title of the book 'A Total Strategy Against Cancer – In Theory and Application' was chosen by Shri Debesh Roy, Prof. Subir Dutta and Shri. Pushpendra Bhowmik. Prof. Subir Dutta and Prof. Jaydeep Biswas had time and again assured me that the module of my work got documented though this initiative. Again this book 'The Management of Cancer in Totality – India can take a lead' aims at opening a new horizon in the treatment and management of cancer.



Book Fair - 2014

I would like to acknowledge here the untiring efforts of Mrs. Soma Das, Mr. Shantanu Das, Mr. Somnath Paul, Mr. Shankar Singh and others. Our pet cat Rakhal also kept me inspired. Again the role of Dr. Hiranmoy Mukherjee deserves special mention. He relentlessly acted as my mentor and advisor on many fronts.

A FEW CASE STUDIES

Cancer has become a threat to human civilization over the centuries since its introduction in the medical scenario. It not only affects the patient but scared the whole community including relations, friends and physicians. Now-a-days various method of diagnosis like F.N.A.C., biopsy, PET Scan etc. are available and early detection of the disease is achieved but it affects the patient and his or her family considerably in terms of expenditure and moreover the expenses incurred in Chemotherapy, surgery, Radiotherapy is also very high. Despite all sorts of investigations and management, the prognosis is on many occasions misleading or hypocritic.

In this background the application of 'Psorinum Therapy' at the terminal stage of the patient concerned throws some light in the direction of effective management of the disease. It is seen that in many cases it not only prolongs the life span of the patient but also relieves him/her from pain.

It is also interesting to note that this therapy has no interaction with other supportive therapy. Psorinum Therapy may be conducted along with the treatment of other underlying diseases like diabetes and other metabolic diseases, hypertension and coronary artery diseases, peptic ulcer and gastro-intestinal diseases, respiratory tract infection, wound and tropical diseases (including Medical Entomology and Protozoology).

Dr. Hiranmoy Mukherjee

Case no.: 1

A 78 years old patient named Gobindo Mukherjee had been admitted at CCMRCC on 23.11.2014. On 10.9.2014 the CT Scan of the Thorax was conducted which indicated mass lesion in mild thoracic oesophagus with dilated upper oesophagus, central bronchiectasis in both lungs, subtle scarry lesions in both upper lobes. The upper G.I. Endoscopy was done where malignant oesophagus mass was found. Biopsy was performed from the oesophageal mass and the report suggested squamous cell carcinoma. After admission, on 24.11.2014 the patient complained of fever with chill and rigor and his blood tests showed studded with M.T. Rings (P F malaria). The patient was suffering from CA – oesophagus and he could not take solid food (Dysphagia). Under these circumstances he was treated and with injection E-Mal – 1 amp 1m X 3 days, followed by a course of Lumether Forte – 1 tablet twice daily. To kill the gametocytes Primaquin 6 tablets was given but the patient could not swallow it. So he was kept in a mosquito net to prevent that spread.

He became febrile and his blood slide did not show any M.T. Ring in the course of the oral treatment and gametocytes disappeared after two weeks.

The patient died on 21.03.2016 due to some elderly disease. So it may be concluded that a synergistic system of treatment which includes modern medicine with Psorinum therapy is achieved with success.

Reports and supporting documents of Gobindo Mukherjee

BINAYAK IMAGING & DIAGNOSTIC PVT. LTD.
59, KALICHARAN GHOSH ROAD, KOLKATA-50
Tel: 9836411177, Email ID : binayakhealthworld@yahoo.co.in

Patient Name	GOBINDA MUKHERJEE	Age / Sex	78/M
PatientID	091/442 ENDO	Visit Date	26/08/2014
Examiner :	DR SANJAY BANERJEE	Ref.Doctor :	DR A K DAS

UPPER GI ENDOSCOPY

PROCEDURE : UPPER G.I. ENDOSCOPY

Instrument: Olympus GIF Q150 Video Scope

Anaesthesia: Xylocaine 2% jelly

Indication:

FINDINGS

OROPHARYNX: Normal

ESOPHAGUS: G E junction at 40 cm from incisor teeth
Lobulated polypoidal mass arising from esophageal wall
starting at 29 cm upto 34 cm with partial luminal
narrowing

STOMACH: Contents: nil; distensibility: good

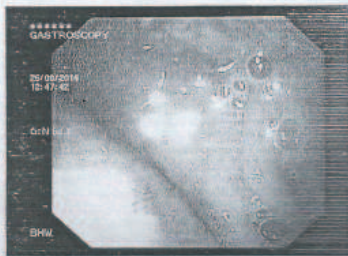
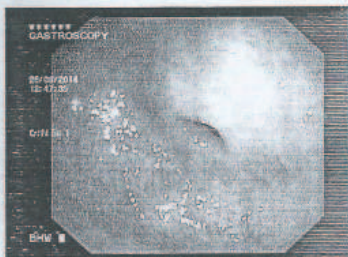
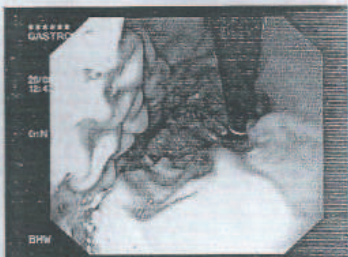
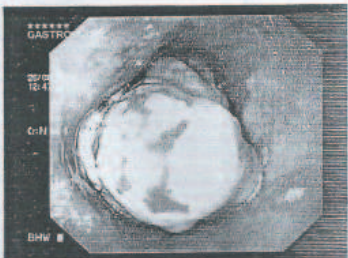
Fundus : Normal mucosa
Body : Normal mucosa
Antrum : Multiple scattered erosion seen
Pylorus : Normal mucosa

DUODENUM: Seen upto D2
First part : Normal mucosa
Second part : Normal mucosa

BIOPSY : taken

IMPRESSION : ESOPHAGEAL MASS --- ? Malignant

DR. SANJAY BANERJEE
MD, DNB, DM(Gastro)
CONSULTANT GASTROENTEROLOGIST



PATIENT NAME :	MR GOBINDA MUKHERJEE	REG. DATE :	26/08/14
AGE	78 YEARS	SEX	M
REF. BY :	DR. A K DAS	REPORT DATE:	07/09/14
		REG. NO. :	H 26/81

REPORT ON HISTOPATHOLOGY
No. BHW:280/14

SPECIMEN :

Endoscopic biopsy from polypoidal mass arising from esophageal wall.

GROSS:

Tiny bits tissue. All embedded.

MICROSCOPY


Section shows tiny fragmented bits of tissue with crushing artifact. There is dense infiltration of mixed inflammatory cell and degenerated cells. Few atypical squamoid cells with degenerative changes are seen within the inflammatory exudate.


IMPRESSION:-

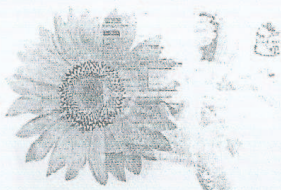
The features are suggestive of squamous cell carcinoma.

*Advice : Correlation with endoscopic features.
 Repeat more representative biopsy may be considered.*

ENCLOSED : 01 Slide.


 DR. ANUP KR. BOLER
 MD (Path)
 Asso. Prof NRS Medical College Kolkata
 Consultant Pathologist


 DR. MANISHA SARKAR
 MD
 Consultant Pathologist



Name	: GOBINDA MUKHERJEE	Age: 78 Years	Gender: Male
Lab No.	: 210210376	Registered: 10/09/2014	
Referred by	: MAJOR DR. SHIBASHISH BHATTACHARYA	Reported : 10/09/2014	

CT SCAN OF THORAX

Procedure :: Plain and Non-ionic contrast enhanced helical / HR CT scan of Chest was done by taking few 10/1 mm. thin slices in upper, mid and lower zone.

Digital Radiography :: Digital radiography of Chest in supine frontal projection reveals no detectable abnormality.

Findings ::

Chest wall : Bony thorax and overlying soft tissue are within normal limits.

Pleura : Pleura is within normal limits without any effusion, thickening nor any nodular and calcific lesions.

Mediastinum : Mediastinal vascular structures and its surrounding fat planes are normal. There is no hilar lymphadenopathy. Trachea, right and left main bronchi, subcarinal, prevertebral, retrosternal & aorto-pulmonic regions are within normal limits. Poorly enhancing soft tissue density mass lesion is seen in mid thoracic esophagus, starting from subcarinal level and measures 8.2 cm. in length and 3.2 cm. in width. Fat planes with the adjacent mediastinum is preserved. G. E. junction is within normal limit.

Lung : Subtle scarry lesions are seen in both upper lobes, anterior segments. Central bronchiectasis is seen in both lungs – predominantly in lower lobes. No obvious focal parenchymal lesion is seen.

IMPRESSION :

- Mass lesion in mid thoracic esophagus with dilated upper oesophagus.
- Central bronchiectasis in both lungs.
- Subtle scarry lesions in both upper lobes.

Am
DR. SANJIB MAJUMDAR
MD (Radiodiagnosis)

Note:

1. This is only a professional opinion based on interpretation of various images and not the final diagnosis.
2. The findings have to be correlated with clinical and other investigations.
3. In case of any discrepancy, please contact the Laboratory immediately.
4. Report/Opinion is not valid for medico legal purposes.

This finding is only arriving at a diagnosis in conjunction with clinical presentation & related investigations as referral guidance. Surgery should not be undertaken only on the basis of this opinion. Several factors like individual assay procedure, method specificity & sensitivity, equipments & reagents / chemicals used and quality of specimen(s), as applicable can cause significant variation. Technical information in this report should be reviewed by qualified medical practitioners only. Please contact us for clarification and for further opinion / advice. Patient identity is not verified in case of pathology samples collected from outside.



R B Diagnostic Pvt. Ltd.

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R B Scan Centre (MRI-CT Unit)
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rbdiagnostic@gmail.com
www.rbdiagnostic.com

ACC. NO. : DH6-209/MRI-24
NAME : Mr. GOBINDO MUKHERJEE
AGE / SEX : 80 Year(s) / Male
REFERRED BY : Dr. ASHIM CHATTERJEE

DRAWN : 06.08.2015 06:14 PM
RECEIVED : 06.08.2015 06:14 PM
REPORTED : 07.08.2015 07:05 PM
CENTER : A-OPD

C.T. of THORAX (Plain & Contrast)

Procedure :

Digital skiagram in AP projection in supine posture does not show any abnormality.

Findings :

The study reveals expansile lobulated sort of soft tissue mass in the middle oesophagus extending from the carinal region and involving approx. 7cm long segment of esophagus. It is hypodense in density. In contrast study inhomogeneous enhancement is seen. There is associated luminal narrowing & shoulder effect is seen at proximal and distal ends of SOL (seen in sagittal and coronal images). There appears loss of plane between it and adjacent aorta & smooth impression over the main bronchi inferior to carina. However no definite aortic or bronchial involvement is seen. There is associated proximal esophageal dilatation.

Lung parenchyma is normal in density on both sides. No significant focal lesion is seen on either side except few fibrotic opacities in right apical region.

Mediastinal vascular structures appear normal. Trachea and main bronchi also appear normal. No significant enlarged lymph node is seen.

No pleural effusion is seen on either side.

No obvious chest wall related lesion is seen. Note is made of degenerative changes in spine.

Cont p/2

This laboratory finding is only in arriving at a diagnosis in conjunction with clinical presentation & related investigations as referral guidance. The reported results are dependent on individual assay procedure, method, specificity, sensitivity, equipments used and quality of specimen(s) received, as applicable. This investigation report is not valid for medico legal purpose. For any kind of technical queries relevant to this report, please contact us immediately.
For pathology sample collection & report delivery at your doorstep, Call : 2521 8888, 4007 5888. You may avail report on e-mail

ACC. NO. : DH6-209/MRI-24
 NAME : Mr. GOBINDO MUKHERJEE
 AGE / SEX : 80 Year(s) / Male
 REFERRED BY : Dr. ASHIM CHATTERJEE

DRAWN : 06.08.2015 06:14 PM
 RECEIVED : 06.08.2015 06:14 PM
 REPORTED : 07.08.2015 07:05 PM
 CENTER : A-OPD

Page 2

IMPRESSION :

CT shows fairly large SOL in middle esophagus with luminal narrowing and proximal esophageal dilatation as described above – suggestive of Ca esophagus.

No other significant abnormality is seen in thorax except few fibrotic opacities in right apical region.

Suggested clinical correlation and further investigation, if indicated.


DR. VIJAY GUPTA, MD
 Consultant Radiologist

Machine Details:
 Spiral CT scan 16 slice Bravo 385
 Low radiation dosage technology.

----- End of Report -----

Case no.: 2

Bhabani Ghosh, a 65 year old lady, was suffering from stomach pain and vomiting. She was admitted to the Ramkrishna Mission Seva Pratisthan on 10.4.1998. The surgery of the gall bladder and the liver metastitis was conducted. The patient was advised to undergo Chemotherapy. As the general condition of the patient was not good, so Chemotherapy could not be administered. The multiple sections studied showed features of a moderately differentiated papillary Adenocarcinoma. The tumour was infiltrating transmurally and was seen in the serosal fat. Ascietis had also developed. The Psorinum Therapy was applied from 2.6.1998. Within the first six months of treatment the ascietis and the liver SOL totally regressed.

The patient was going well till 2012. On 17.9.2012 the whole abdomen CT Scan was conducted. CT features suggested Pneumobilia, smaller right kidney and omental thickening at places. After this no more follow-up was done. After some months we came to know that the patient had passed away.

Reports and supporting documents of Bhabani Ghosh

Serial No. 2061

Ramakrishna Mission Seva Pratisthan
99, Sarat Bose Road, Calcutta-700 026
HISTOPATHOLOGICAL REPORT

Name Bhabani Ghosh Sex F Age 65
M.R.D. No. 3427 Ward 2 Bed/Cabin 2-4 OPD Reg. No. 673/98

Gross: - 2 cm (1) small tissue bits from Gall Bladder

Micro - Multiple section studied show features of a Moderately differentiated Papillary Adenocarcinoma. The tumour is infiltrating transmurally and is seen in the serosal fat

Tag - Gall Bladder → Moderately differentiated Papillary Adenocarcinoma

17/4/98

Signature of Specialist

EKO CT & MRI SCAN CENTRE



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(A Joint Venture with Eko Diagnostic Pvt. Ltd. &
Dept. of Health & Family Welfare, Govt. of West Bengal)

BHABANI GHOSH

72 YRS

17.09.2012

DR. OF MCH

13921/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

Follow up patient of Ca. gall bladder.

TECHNIQUE

Plain, oral and I.V. (non -ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no detectable abnormality.

Liver is normal in size – shows pneumobilia.

Gall bladder is operated.

Common bile duct is not dilated.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Kidneys : Right kidney is shrunken in size. Left kidney shows dilated pelvis.

Contd ...2..

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Dept. of Health & Family Welfare, Govt. of West Bengal)

-2-

BHABANI GHOSH

72 YRS

17.09.2012

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Uterus is bulky.

Adnexal regions are clear.

Omental thickening seen at places.

Lung bases are clear.

IMPRESSION


CT features are suggestive of :

- 1) Pneumobilia.
- 2) Smaller right kidney.
- 3) Omental thickening at places.

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD


DR.ANUP SADHU
DMRD, MD.

Case No. 3

Mr. Swapan Kumar Majumdar, a 40 year patient, complained of pain in the left waist for six months and patient was suffering from swelling in the right side of neck for 12 years and chronic cough for 3 months. Patient was admitted in the hospital in June, 2005 with severe abdominal pain. On 30.5.2005, CT Scan findings of upper abdomen suggest a well-defined heterogeneously enhancing lesion with peripheral calcifications in the right peri-aortic region and another large heterogeneously enhancing lesion was noted in the left suprarenal gland favouring neo-plastic conditions. On 7.6.2005, FNAC report of the right upper deep cervical lymph-node yielded suspicions of metastatic carcinomatous deposit. Patient was advised for CT Thorax, and USG of whole abdomen. On 22.6.2005, CT Scan findings of thorax were suggestive of old KOCH's scar at both apical region of upper lobes with ill-defined nodular infiltrations in left lower lobe. On 23.6.2005, USG of whole abdomen suggested – large left adrenal mass and enlarged (metastatic) lymph-node between IVC (Inferior vena cava) and aorta at level of origin of renal vessels.

Patient's health condition deteriorated to such an extent that any kind of movement was impossible. He was totally bed-ridden. Patient did not undergo any RT and CT Scan. Since June, 2005 patient underwent the treatment of Psorinum Therapy along with supportive palliative. Subsequent CT Scan on 18.11.2005 of upper abdomen for review study shows almost identical size of abdominal lesions as described since last study of 30.5.2005. On 24.7.2006 CT Scan of upper abdomen was compared with the previous CT dated 18.11.2005, the appearance was almost similar. Another CT Scan of the chest suggested no obvious abnormality in the thorax. Dr. Jaydeep Biswas treated this patient since 15.9.2006 and the patient's case was registered in Chittaranjan National Cancer Institute (CNCI) Hospital. The patient is now leading a quite simple life and runs a stationary shop. Till August 2012, he continued his Psorinum Therapy at our Critical Cancer Management Clinic & Research Centre.

Reports and supporting documents of Swapan Kumar Majumdar

EKO DIAGNOSTIC

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Fax No. : 282-8098
E-mail : ekoxray@satyam.net.in



DIVISION OF IMAGING

MR SWAPAN MAJUMDER

40 YEARS

30.05.2005

DR A-K ACHARYA

CT SCAN OF UPPER ABDOMEN

HISTORY

Uneasiness in left upper abdomen. Loss of appetite. USG revealed a left adrenal mass.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced MDCT scan of the upper abdomen done in the axial plane in spiral mode followed by multiplanar reformations.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no significant abnormality.

Liver is enlarged in size and normal in shape, position, outline and density. The intrahepatic biliary radicles are not dilated. No focal lesion is detected. Portal vein appears normal.

Gall bladder appears normal and its lumen does not show any radio-opaque calculus or intraluminal lesion (However, radio-lucent and small calculus may be missed in C T. USG may be done for further evaluation).

Common bile duct is not dilated.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Contd....2..

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DIVISION OF IMAGING

2

MR S MAJUMDER

(CT OF UPPER ABDOMEN)

30.05.2005

Right suprarenal gland appears normal in size, morphology and density. No evidence of nodularity or SOL is detected on the right side. Left suprarenal gland is not clearly visualized.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal. Both upper ureters show normal course and calibre.

Aorta and IVC are normal. Well-defined heterogeneously enhancing lesions are seen in the right peri-aortic region with peripheral calcifications measuring about 3.5 cm. x 2.6 cm. in the maximum axial dimension (Vide Series 3, Image 34) and another larger one in the left suprarenal gland region measuring about 6.5 cm. x 6.2 cm. in the maximum axial dimensions (Vide Series 3, Image 22). No free fluid is seen in the peritoneum.

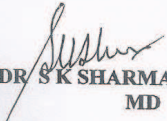
Bones under review show no detectable abnormality. Parietal and paravertebral muscles including psoas muscles are normal.

IMPRESSION


MDCT features suggest a well-defined heterogeneously enhancing lesion with peripheral calcifications in the right peri-aortic region and another larger heterogeneously enhancing lesion is noted in the left suprarenal gland favouring neoplastic conditions - ? lymph nodes vs. any other pathology.

Hepatomegaly is also present.

Suggested clinico-pathological correlation and other investigations for further evaluation and confirmation.


DR S K SHARMA
MD


DR S ROY
MD


DR B KUNDU
DMRD

DR S KUNDU
MD

EKO PATHOLOGY CENTRE



(HISTOPATHOLOGY / CYTOLOGY / FNAC)

DR. SHYAMALENDU MANDAL

MBBS (CAL) MD(PATH) PGI(CHANDIGARH) MIAC
EX-SENIOR REGISTRAR - A.I.I.M.S. (NEW DELHI)
CONSULTANT HISTOPATH & CYTOPATHOLOGIST (FNAC)

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E-mail : ekoxray@satyam.net.in

MR. SWAPAN MAJUMDAR

Age : 40yrs.

Dt. of receipt : 30.05.05

REFD. BY DR. A. K. ACHARYA

L4055

Dt. of report : 01.06.05

FNAC REPORT

(FN/453)

Site of aspiration :

Left suprarenal space occupying lesion(SOL)

Gross examination :

CT guided FNAC of the left suprarenal SOL yielded particulate material.

Microscopic examination :

Smears show multiple clumps and scattered atypical cells having round mildly pleomorphic hyperchromatic nuclei with clumped chromatin, prominent nucleoli and abundant vacuolated cytoplasm on the mild haemorrhagic background.

Diagnosis:

Adrenocortical carcinoma.

Note:

A histopathological examination is necessary for confirmation.

Slides enclosed:

2

DR. SHYAMALENDU MANDAL M.D.
Chief consultant

Quadra Medical Services Pvt. Ltd.

41, Hazra Road, Kolkata - 700 019 Phone : 2474-1820 / 1821 / 4455 / 4466

Page No. 1 of 1
Report No. F060333 Printed on : Tuesday, June 7, 2005

=====
Code__ : F060331 Name__ : SWAPAN MAJUMDER Sex__ : Male Age__ : 40
Ref. Doctor__ : Q.M. RAHMAN / A. ACHARYA
=====

REPORT ON FNAC

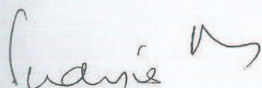
1468/05

FNA of the right upper deep cervical lymphnode yielded particulate material.

Smears are moderately cellular. They show a population of round cells arranged in microacinar pattern, clusters or discretely admixed with lymphoid cells. The round cells show mild anisonucleosis with some of the cells showing prominent nucleoli.

DIAGNOSIS : The overall cytomorphological features are suspicious of metastatic carcinomatous deposit.
Please correlate with the clinical features.
Histological examination may be considered for confirmation.

* Slides provided.



DR SUDIPTA ROY
MBBS, MD (PGI), DNB, DRCP (UK)
Chief of Pathology

Prepared By : AB
Printed By : AB

EKO NUCLEAR IMAGING CENTRE



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MR SWAPAN MAJUMDAR

40 YEARS

09.06.2005

DR Q M RAHMAN

BS1006

RADIONUCLIDE WHOLE BODY BONE SCAN:

HISTORY

Metastatic CA.

STUDY

Whole body bone scan in anterior and posterior views and spot view of RAO thorax & LPO thorax were obtained in Gamma Camera after I. V. injection of 20mCi 99mTc-MDP.

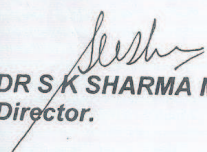
FINDINGS

No significantly abnormal concentration of radiotracer is observed in the skeleton either in the anterior or in the posterior views.

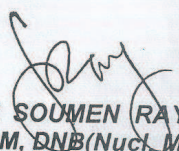
Both kidneys are visualized.

IMPRESSION

NORMAL BONE SCAN.


DR S K SHARMA MD
Director.

DR NILRATAN DEY
DRM, Consultant


DR SOUMEN RAY
DRM, DNB(Nucl Med)
Chief Consultant.

- | | | | |
|-----------------------------------|--------------------|------------------------------|------------------------|
| ● BONE SCAN | ● THYROID SCAN | ● RENOGRAM | ● LIVER SCAN |
| ● THALLIUM SCAN | ● MYOVUE SCAN | ● MIBI SCAN | ● HEPATOBILIARY SCAN |
| ● MUGA | ● GI BLEEDING SCAN | ● MECKEL'S DIVERTICULUM | ● GE REFLUX STUDY |
| ● BRAIN SPECT | ● GALLIUM SCAN | ● PARA THYROID SCAN | ● RADIO-IODINE THERAPY |
| ● MIBG SCAN | ● TESTICULAR SCAN | ● ISOTOPE VENOGRAM | ● METASTRON THERAPY |
| ● VENTILATION PERFUSION LUNG SCAN | | ● SPLENIC SEQUESTRATION SCAN | |

Dr. Jaydip Biswas

MBBS, MS, DMRT, FAIMS, FAIS, FICS
Consultant Surgeon & Oncologist

Date 22/06/05

Ref.

Mr. Swapan Mazumder
40 yrs M.

1/0 Pain in the @ loin
for @ months.
Swelling in the @ side
of neck for 12 yrs.
↑ for last 3 months.
Chronic cough for 3 months.

F.N.A.C. for this @ Cervical node.
Suspicious of metastatic
Concomitant deposit

C.T. Scan abdomen.
L.R. @ Suprarenal gland
? lymph node vs. any other
pathology -

24-hr VMA - 45.01 ng/g creat

O/E G.S. - good G.S. BP 110/70 mm Hg.

@ Cervical lymph node
No other lymphadenopathy
Chest - Unremarkable Bx vague mass in @ loin.

Adh
• C.T. Thorax

• Abroad for TC & US?
Platelet count
BT/CT.

Super < E
PP

L.F.T.

WBC / uric acid.

• U.S.G. whole abdomen
• urine for VMA (repeat)
↓

Rem with his
reports.

A
22/06/05

Cap Antegrade path
1 Cap Ad

Eng. Lab. for long 1 h.

Residence : EE-174 A/2 (Near 10 No. Tank Petrol Pump), Salt Lake City, Kolkata - 700 091
Phone : 321-9878, Mobile : 98300 26696, E-mail : drjaydip_biswas@yahoo.com

A
22/06/05

and photo

No fix. in between
Metastatic

① Cervical lymph node
in a can of

Adrenocortical CA(L)
with Loz in adrenal
gland.

Adm • Dr. S. G. Meek to
see the patient & discuss & by record.
• To attend any Medical
College Hospital, Kolkata
for institutional
management.

A 10/9/06

~~R. S. G. Meek~~

Study Case

CNCI
CHITTARANJAN NATIONAL CANCER INSTITUTE
HOSPITAL

37, S. P. MUKHERJEE ROAD, KOLKATA - 700 026, INDIA

HISTOPATHOLOGY FORM

Name Swapna
Aditya Kumar Mukherjee Regd. No. 5/05/4212 Bed No. 8090

Age 42 yrs Sex M Religion H

Referred from Department Consultant

Short Clinical History Pain in the Lt. lumbar region.

Nature of Examination required slide review

Slide No. 5

Date 15/9/06

To path

15/9/06
 Signature of Medical Officer

REPORT

Received By 10/10/06
 5/10/06

Ref 525/06 5/10/06
Subj: Adrenal

Slide review. F/453

CT Guide FNA from the left Suprarenal Soc.

Microscopic: Moderately cellular smear, clusters of cells with hypochromatic pleomorphic nuclei, homogeneous chromatin and prominent nucleoli. Cytoplasm is broad fine there is a tendency of acinar formation. There are plenty of nuclei with prominent nucleoli in the background.

Imp:

Features are consistent with Adrenocortical carcinoma. However, Pheochromocytoma comes in close D/D.

Please corroborate with CT, USG, & VMA level.

Slide no. 1468/05 - FNA from deep cervical lymph node.

Microscopic: Moderately cellular smear, clusters of cells with broad cytoplasm, pleomorphic nuclei with prominent nucleoli. There is a tendency of acinar and papillary formation. Background Blood.

Imp:

Features are consistent with metastatic adenocarcinoma.

Kanwal PATHOLOGIST

Case No. 4

Chondrosarcoma is a rare form of bone tumour. It develops from normal cartilage which goes through malignant change, or can form within a pre-existing benign tumour called osteochondroma or enchondroma. People who have chondrosarcoma have a single tumour growth which can vary in size and location. Malignant bone tumour spread to other tissues and organs and are on most of the occasion life-threatening. Chondrosarcoma is not the type of bone cancer that spreads from other organs to the bone. That is called metastatic bone cancer which might be located in more than one bone. A typical example is when lung, kidney, liver, breast or other cancer spreads to the bones as part of their metastasis.

While treating cancer in different organs of the body, on many occasions the disease spreads to the lungs. Such cases are very difficult to manage specially in the case of sarcoma where chemotherapy or radiotherapy cannot be administered and surgery is the only alternative left in the case when the affected organ is operatable. In such cases surgery can be performed more than once. We have observed that in these kinds of cases Psorinum therapy is found to be very effective. I would like to discuss a case here:

Dipankar Chakraborty, a citizen of Bangladesh, Dhaka, and brother of Advocate Gautam Chakraborty (former Minister of Water Resources, Bangladesh) was a case of Chondo-sarcoma in 2004. He had tumour in right bone. In 2007 he suffered from chest pain and cough. In the same year, he was diagnosed by Prof. (Doctor) Mayil Vahanan Natarajan. Under his supervision, chest scan report showed multiple bilateral parenchymal nodules. Also multiple metastases were found in both of his lungs. Mostly these types of Cancer are radiotherapy and chemotherapy resistant. Before coming to us the patient had undergone surgery a few times from 2004 –07. The patient visited us in October 2007 and since then the patient is under Psorinum Therapy. We could successfully arrest the growth of the disease and for the past nine years the condition of the patient is quite stable. The last Chest scan report of 16.11.2012 showed identical appearance. This case was jointly handled by Prof. Jaydeep Biswas, Dr. Aradeep Chatterjee and I.

Reports and supporting documents of Dipankar Chakraborty



PEERLESS HOSPITAL & B. K. ROY RESEARCH CENTRE

Regd. Office : 360 Panchasayar, Kolkata-700 094, Phone : 2462-2394/2462/0071-73, Fax No. : 0091-033-2462-0766
E-mail : hospitex@vsnl.com, Website : www.peerlesshospital.com

ISO 9001 : 2000

DEPARTMENT OF PATHOLOGY

HISTOPATHOLOGY BIOPSY (MEDIUM)

Patient Particulars :

Patient no : 224006461 Diagnostic serial no : 724194419
Room No 2029 Bed No : 2029 Patient's Name : DEPONGKAR CHAKRABORTY
Age : 43 Yrs Sex : M Date : 08/11/2004 16:55
Referred By : Dr. PRABIR K. BANERJI
Print date : 09/12/2004 16:11

SPECIMEN	PARTS OF TUMOUR TISSUE (R) ILIUM REGION.
GROSS	THREE IRREGULAR TISSUE PIECES LARGEST 2.0 X 2.0 X 1.0 CM. REPRESENTATIVE SECTIONS TAKEN.
MICROSCOPIC EXAMINATION	SECTIONS SHOW LOBULATED MYXOID AREAS, SOME DEVOID OF ANY CELLULAR MATERIAL. THE OTHERS SHOW SMALL ROUNDED TO ELONGATED CELLS ARRANGED IN ANASTOMOSING CORDS. THE CELLS HAVE EOSINOPHILIC CYTOPLASM, SMALL VESICULAR NUCLEI AND SMALL NUCLEOLI. TOWARDS THE PERIPHERY THE CELLS ARE MORE IN NUMBER. NO MITOTIC FIGURES SEEN. THE TUMOUR ARE INFILTRATING ADJACENT MUSCLE FIBRES.
IMPRESSION	MYXOID CHONDROSARCOMA. IT IS A <u>LOW GRADE MALIGNANT TUMOUR</u> WITH <u>HIGH RECCURRANCE RATE.</u>
SLIDE NO	2278/04

Dr. DILIP KUMAR ^{mitra} MITRA
DCP, D.R.C.PATH. F.R.C.PATH(ENG)
CONSULTANT HISTOPATHOLOGIST.



ISO 9001 : 2000

PEERLESS HOSPITAL & B. K. ROY RESEARCH CENTRE

Regd. Office : 360 Panchasayar, Kolkata-700 094, Phone : 2462-2394/2462/0071-73, Fax No. : 0091-033-2462-0766

E-mail : hospitex@vsnl.com, Website : www.peerlesshospital.com

DEPARTMENT OF PATHOLOGY

HISTOPATHOLOGY BIOPSY (MEDIUM)

Patient Particulars :

Patient no : 624037036

Diagnostic serial no : 724215081

Patient's Name : DIPANKAR CHAKRABORTY

Age : 46 Yrs Sex : M

Date : 04/12/2004 10:57

Referred By : Dr. P K BANERJEE

Print date : 09/12/2004 11:16

SPECIMEN

TUMOUR TISSUE SLIDE REVIEW.
(R) ILIAC REGION.

GROSS

STAINED SLIDES RECEIVED FOR REVIEW.

MICROSCOPIC EXAMINATION

ALL THE SECTIONS SHOW FAIRLY WELL CIRCUMSCRIBED LOBULES OF MYXOID MATERIAL AMONGST GROUPS OF STRIATED MUSCLE. IN THE POOL OF MYXOID MATERIAL THERE ARE SMALL ROUNDED AND ELONGATED CELLS. THE CELLS HAVE UNIFORM NUCLEI AND THE CYTOPLASM IS EOSINOPHILIC. NO MITOTIC FIGURES SEEN.

IMPRESSION

MYXOID CHONDROSARCOMA.

SLIDE NO

2412/04

Dr. DILIP KUMAR MITRA
DCR, D.B.C. PATH. F.B.C. PATH. (ENG)
CONSULTANT HISTOPATHOLOGIST.



ANOWARA DIAGNOSTIC CENTRE

44/12 West Panthapath (Opposite Samorita Hospital)
Dhaka, Bangladesh. Phone: 9123253, 9145908

ULTRASONOGRAM GUIDED FNAC REPORT

Patient's	: Mr. Dipankar Chakraborty	Patient's ID	: 000000084591
Age	: 47 Year(s) Sex : Male	Case ID	: 060000008645
Referred by	: Dr. Md. Mokles Uddin FCPS Radiotherapy		
Specimen	: Iliac fossa mass right	Lab No	: 06-003-2142
Investigation	: USG Guided FNAC	Exam Date	: 11 May 2006

Clinical Information

Ultrasonogram Findings : Ultrasonogram of abdomen is done. A hypoechoic lesion is seen in the right iliac fossa.

Aspiration Note : A needle is introduced. The needle tip is identified within the lesion. Aspirated a few drops of haemorrhagic material. No immediate complication is seen.

Microscopic Description

Comment : Smear shows adequate cellular material containing malignant round cells in the background of myxoid material. The round cells are compatible with chondroid material.

Diagnosis

Dx : Iliac fossa mass right (USG guided FNAC) : Compatible with myxoid chondrosarcoma (See Comment).

With Compliments for kind referral

Mohammad Golam Mostafa
(Dr. Mohammad Golam Mostafa)
MBBS, M Phil Pathology (Honors), MIAC
WHO Fellow in Cytopathology, Bangkok
Professor (C.C.) of Histopathology
National Institute of Cancer Research and Hospital
E-mail: mostafag@btb.net.bd

আনোয়ারা ডায়াগনস্টিক সেন্টার
৪৪/১২ পশ্চিম পাথপথ, ঢাকা
(শমরিতা হাসপাতালের উল্টোদিকে)
ফোন: ৯১২৩২৫৩, ৯১৪৫৯০৮



Mr. DIPANKAR CHAKRABORTY

[49/M]

26th October 2007

ID.NO.: BS / CT / AN / 20261007

Ref. By: **Prof. Dr. MAYIL VAHANAN NATARAJAN**, M.S.Orth. (Ma's) M.Ch.Trauma (L'pool) Ph.D. (Orth. Onco.), D.Sc.

DUAL SLICE - CT - CHEST

Helical plain CT study of the chest:

Multiple small noncalcific nodules (~ 10mm) with well defined margins noted throughout both lungs, more at the bones.

Mediastinal position and contents including the trachea and its bifurcation, thoracic aorta, the main pulmonary artery and its branches appear normal.

No definite mass lesions identified in the mediastinum / hilar regions.

No significant lymph node enlargement identified.

No significant pleural thickening / fluid collection seen.

No bony destruction seen.

Superficial soft tissues of the chest wall appear normal.

IMPRESSION:

- ◆ **Multiple bilateral pulmonary parenchymal nodules**
- ◆ **Suggestive of secondaries**

Encl. film: 3

Dr. G. Srinivasaraman, DMRD, DNB
Radiologist
Mobile: 98412 80695

Quality is our Image

Corporate Office Royapettah : 197, Peters Road, Royapettah, Chennai - 600014. ☎ : 2841 8334 / 35 / 36 / 37 / 98410 63666
Anna Nagar : AB-1, 11nd Avenue, Anna Nagar, Chennai - 600040. ☎ : 2626 7000 (7 Lines) ✆ 98410 59900
Ashok Nagar : 81, 1st Avenue, (Near Ashok Pillar), Ashok Nagar, Chennai - 600083. ☎ : 2489 4677, 2471 0777, 2474 7772 / 98410 46663
Guindy : S7, Thiru Vi Ka Industrial Estate, Guindy, Chennai - 600032. ☎ : 2232 8118, 2231 1423, 2231 6500 / 98410 62221
www.bharatscans.com



Mobile : 9748453935
Phone : (91) 033 2534-4702

Critical Cancer Management Research Centre & Clinic

(Run by Dr. Ashim Chatterjee & Other Renowned Oncologist & Onco Surgen)
381, S. K. DEB ROAD, KOLKATA-700 048

Ref. No. _____

Date 6/5/08

Rec. case of
Mucoid Chondrosarcoma
"Rt iliac bone".
CT thorax → Metastasis.

Mr Dipankar Chakraborty
49 yrs/M

Adm

① CT Scan thorax [Please compare w the
previous CT thorax].
E continued

② U.S.G Wt abdomen

In exam

C.S. good

C.S. NDD

Chest → clear

Abdomen → Lm
Apr. 11/08

Two masses -

- one in Rt iliac bone
- one in soft medulla

with peritoneal
involvement

① bilateral abdominal
above ② iliac
mass

Plan for
J treatment

Signature
6/5/08

Dr. Ashim Chatterjee

9830026696

94330-26696

Signature of Visiting Consultant



NORTH CITY

* DEPARTMENT OF IMAGING *

Regd. No. CT 11505

=====

Patient's Name : DIPANKAR CHAKRABORTY	Age : 49 Y	Sex: M
Ref. by : DR. JAYDIP BISWAS	Date of Scan : 07.05.2008	
	Date of Report: 08.05.2008	

=====

* Thank you for kind referral.

CT SCAN OF THORAX

PROCEDURE :-

Axial plain CT scan of chest was done from lung apex through domes of the diaphragm by taking 5 mm thin parallel slices.

DIGITAL RADIOGRAPHY :-

Digital radiography of chest in supine AP view shows sternotomy sutures.

FINDINGS :-

Mediastinum shows normal appearances of the great vessels & heart, trachea-bronchi and oesophagus. No enlarged mediastinal or hilar lymphnodes noted.

The axial cuts for the lung fields are showing multiple small non-calcific nodules of various size being randomly scattered in both lung fields and more numerous in right lower lobe along with few in rest of the lung fields bilaterally. No focal bronchiectatic changes seen. No focal reticular lesion seen on either side. No emphysematous bullae seen.

No pleural thickening or effusion noted on either side.

Chest wall shows no obvious abnormality.

Patient party refused contrast injection due to h/o allergy to food grains and penicillin group of drugs.

IMPRESSION :-

FOLLOW-UP CASE OF MULTIPLE SECONDARY DEPOSITS IN BOTH LUNG FIELDS IN A CASE OF MYXOID CHONDROSARCOMA NOW SHOWING ALMOST IDENTICAL APPEARANCES SINCE THE LAST CT SCAN DONE ON 26/10/2007; IN FACT, NO INCREASE IN SIZE AND NUMBER OF THE LESIONS NOTED IN THE PRESENT STUDY.



DR. S. P. BHOWMIK
BSc (Hons), MBBS (Cal), DMRD (Cal),
SENIOR CONSULTANT RADIOLOGIST.

North City Diagnostic Centre (Pvt.) Ltd. 35A, Canal West Road, Kolkata - 700 004
(Near Gouri Bari Bus Stop) Tel : 2554 6221/6225/2543 2374 Fax : 23211100, E-mail : north_city@rediffmail.com
From 7.00 a.m. To 8.00 p.m. (Mon - Sat) From 7.00 a.m. To 2.00 p.m. (Sunday)

EKO CT & MRI SCAN CENTRE

A UNIT OF EKO DIAGNOSTIC PVT. LTD.

(A Joint Venture with Eko Diagnostic Pvt. Ltd. &
Dept. of Health & Family Welfare, Govt. of West Bengal)



DIPANKAR CHAKRABORTY

52 YRS

⁰⁰⁵
16.11.2011

DR. OF MCH

C T SCAN OF THE CHEST

HISTORY

Follow up patient of chondrosarcoma with pulmonary metastasis – previous film not available.

TECHNIQUE

Plain & I.V. (non –ionic) contrast enhanced C T scan of the chest done with 5 mm. and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the chest in supine position and in frontal projection shows no detectable abnormality.

Bones under review shows no detectable abnormality. Parietal muscles appear normal. No sizeable mass is detected in the axillae.

Great vessels of the mediastinum including ascending aorta, arch of aorta and its branches, descending aorta, M.P.A. and its branches, S.V.C. and its tributaries appear normal. Trachea and its bifurcation are normal. No sizeable mass is detected in the mediastinum.

Pulmonary nodules (5-12 mm) seen in both lungs.

No evidence of any pleural thickening or pleural effusion is seen.

Contd2..

CT & MRI SCAN CENTRE

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Joint Venture with Eko Diagnostic Pvt. Ltd. &
(Dept. of Health & Family Welfare, Govt. of West Bengal)



-2-

DIPANKAR CHAKRABORTY

52 YRS

16.11.2011

IMPRESSION

CT features are suggestive of metastatic pulmonary nodules of varying sizes (5 –12mm).

Suggested clinical correlation and further investigations if clinically indicated.

A handwritten signature in blue ink, appearing to read "Anup Sadhu", is written over the printed name of Dr. Anup Sadhu.

DR.S.K.SHARMA
MD

DR.D.SHARMA
MD

DR.ANUP SADHU
DMRD, MD.

Case No. 5

Sumita Paul, a 35 year old lady, came to us complaining severe abdominal pain, jaundice, vomiting and inability to take food. She was advised to undergo ERCP. The report suggested: Duodenal ampulla shows a nodular tumour. A large peri-ampullary diverticulum seen. Cannulation of the bile duct carried out. Common bile duct shows dilation upto lower end. Hepatic ducts and intrahepatic biliary radicles are dilated. No obvious filling defect due to stones seen in the distal bile duct. Ampullary biopsy taken. Endoscopic biliary stenting done. In the histopathology report the microscopic examination and diagnosis indicated: Invasive adenocarcinoma. Moderately differentiated in present biopsy.

The patient was admitted here on 22.04.2014. Effective 03.05.2014 symptomatic allopathic management was also started. Here instead of Chemotherapy Psorinum therapy was administered from the very next day. The next 1.5 years the patient used to come for regular follow up. The patient's condition had drastically improved over the months. But suddenly the patient discontinued the treatment and was not in touch for near about 6 months. But again on 25.4.2016 the patient had to be admitted here following abdominal pain and inability to take food. The CT scan of the abdomen was performed and CT features were suggestive of: Peri-ampullary Ca. (13mmX13mm). Hematomegaly with focal dilation of IHBR at places. However, the patient's current blood picture was quite satisfactory. The medical board decided upon triple by-pass which was later conducted from SSKM Hospital under the supervision of Prof. S. Saha. Psorinum therapy has been started all over again and the patient is presently doing quite well.

Reports and supporting documents of Sumita Paul

CENTRE FOR DIGESTIVE DISEASES

(DIAGNOSTIC AND THERAPEUTIC G.I. ENDOSCOPY)



EKO GROUP

54, Jawaharlal Nehru Road, Kolkata-700 071

☎ : 91 33 2282-0636/8105/06/09/0751 Fax : 91 33 2282-8098
E-mail : enquiry@ekoxray.com Website : www.ekoxray.com

Dr. Mahesh K. Goenka

Consultant Gastroenterologist, Endoscopist & Hepatologist,
M.D. (Med), D.M. (Gastro) MNAMS (Gastro),
FACG (USA), FASGE (USA)
Secretary, Society of GI Endoscopy of India
Fellow, American Society for G.I. Endoscopy
Fellow, American College of Gastroenterology
Member, American Gastroenterology Association
Adjunct Professor, Queensland University, Australia
Formerly Asst. Prof., P.G.I. Chandigarh
Registration No. 41065 of WB Medical Council

ENDOSCOPIC BILIARY STENTING

REPORT OF E. R. C. P. (ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATOGRAPHY)

NAME : MRS. SUNITA PAUL AGE : 35YRS SEX : F

REF. DR : OF BELUR S. S. SAMITY ED06 DATE : 07.04.14

ANASTHETIST DR. : SANJAY MAHAWAR

E.R.C.P. PERFORMED UNDER OXISCOPE MONITORING USING OLYMPUS VIDEO DUODENOSCOPE TJF – Q150

Sedation used : I. V. Propofol
Indication : CBD stone (?)

DUODENUM

Duodenal ampulla shows a nodular tumor. A large peri-ampullary diverticulum seen. Cannulation of bile duct carried out.

BILE DUCT

Common bile duct shows dilatation upto lower end. Hepatic ducts and intrahepatic biliary radicles are dilated. No obvious filling defect due to stones seen in the distal bile duct.

GALL BLADDER

Not filled.

ERCP films provided.

Cont - 2

- | | | |
|-----------------------------------|--|-----------------------------|
| ☆ Video, Gastroscopy, Colonoscopy | ☆ ERCP Variceal Sclerotherapy, Histoacryl, Band ligation | ☆ Foreign Body Removal |
| ☆ Esophageal Endoprosthesis | ☆ Polypectomy | ☆ Stricture Dilatation |
| ☆ Bile duct Stone Extraction | ☆ Biliary Stenting (Metal, Plastic) | ☆ Pancreatic Stenting |
| ☆ Nasobiliary Drainage | ☆ Argon Plasma Coagulation | ☆ Ileoscopy |
| ☆ Capsule Endoscopy | | ☆ Breath test for H. Pylori |

CENTRE FOR DIGESTIVE DISEASES

(DIAGNOSTIC AND THERAPEUTIC G.I. ENDOSCOPY)



EKO GROUP

54, Jawaharlal Nehru Road, Kolkata-700 071

☎ : 91 33 2282-0636/8105/06/09/0751 Fax : 91 33 2282-8098
E-mail : enquiry@ekoxray.com Website : www.ekoxray.com

Dr. Mahesh K. Goenka

Consultant Gastroenterologist, Endoscopist & Hepatologist,
M.D. (Med), D.M. (Gastro) MNAMS (Gastro),
FACG (USA), FASGE (USA)
Secretary, Society of GI Endoscopy of India
Fellow, American Society for G.I. Endoscopy
Fellow, American College of Gastroenterology
Member, American Gastroenterology Association
Adjunct Professor, Queensland University, Australia
Formerly Asst. Prof., P.G.I. Chandigarh
Registration No. 41065 of WB Medical Council

(2)

NAME : MRS. SUNITA PAUL
ED06

PROCEDURE

A 10 Fr. plastic stent positioned in bile duct across the ampulla. Biopsies taken from ampullary area for histopathology examination. No complication seen.

IMPRESSION

- * AMPULLARY TUMOR - ? NATURE.
- ENDOSCOPIC BILIARY STENTING DONE.
- HISTOPATHOLOGY AWAITED.

DR. MAHESH K. GOENKA
Chief Gastroenterologist

- | | | |
|-----------------------------------|--|------------------------|
| ☆ Video, Gastroscopy, Colonoscopy | ☆ ERCP Variceal Sclerotherapy, Histoacryl, Band ligation | ☆ Foreign Body Removal |
| ☆ Esophageal Endoprosthesis | ☆ Polypectomy | ☆ Stricture Dilatation |
| ☆ Bile duct Stone Extraction | ☆ Biliary Stenting (Metal, Plastic) | ☆ Pancreatic Stenting |
| ☆ Nasobiliary Drainage | ☆ Argon Plasma Coagulation | ☆ Ileoscopy |

☆ Capsule Endoscopy

☆ Breath test for H. Pylori



**EKO DIAGNOSTIC
PATHOLOGY**

(a Unit of EKO DIAGNOSTIC PVT. LTD.)

MS. SUNITA PAUL

Age : 35yrs.

Dt. of receipt : 07.04.14

REFD. BY DR. M. K. GOENKA

L000735

Dt. of report : 10.04.14

HISTOPATHOLOGY REPORT

(H/23)

SPECIMEN : Ampullary biopsy

GROSS DESCRIPTION : Single tissue bit, all embedded.

Stains Performed : HE

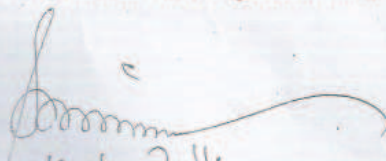
MICROSCOPIC EXAMINATION & DIAGNOSIS :

Invasive adenocarcinoma.

Moderately differentiated in present biopsy.

ADVICE : Clinical / endoscopic correlation.

Slide enclosed: 2


10.04.2014
DR. SWAPAN SAMANTA
M.D (Path), PGI, Chandigarh.

Report relates to the item tested only

All slides included with the report (slide No.)

Blocks will be preserved for 3 months after reporting.

Gross specimen not collected within 15 days will be discarded.

54, Jawahar Lal Nehru Road, Kolkata - 700 071 * Phone : 2282-8106 / 8109 / 0745 / 0715 / 073
Fax : (033) 2282-8098 * E.mail : enquiry@ekoxray.com

EKO ENDOSCOPY

54, Jawaharlal Nehru Road

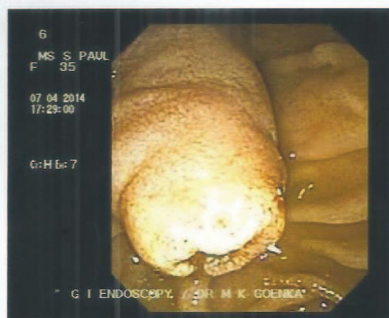
Kolkata – 700 071

DR M K GOENKA

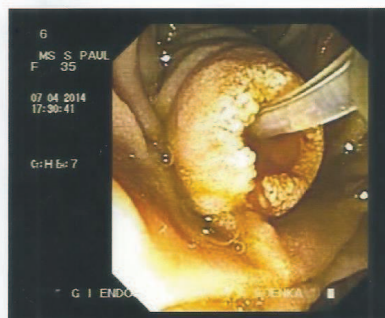
M.D (Med). D.M. (Gastro) MNAMS (Gastro),

FACG (USA), FASGE (USA)

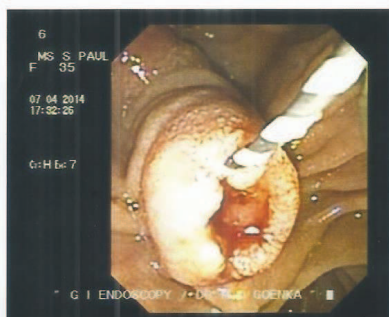
MS S PAUL



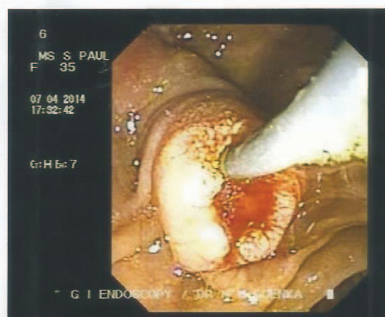
PAPILLA



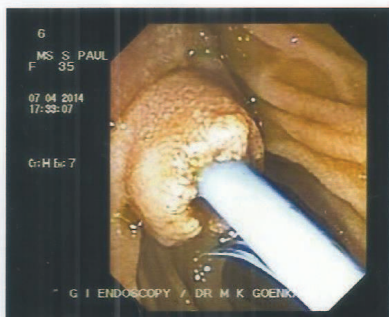
SPHINCTEROTOME



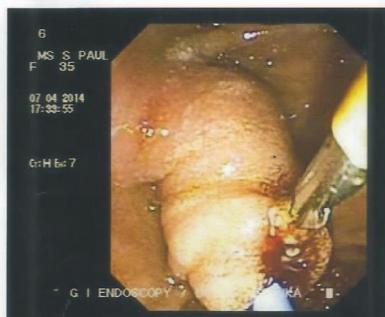
GUIDE WIRE



GUIDING CATHETER



STENT



BIOPSY FROM PAPILLA

Critical Cancer Management Research Centre & Clinic

381, S. K. DEB ROAD, KOLKATA - 700 048

Hospital Registration No.: 1298/N/2008 (Dept. Health & Family Welfare, West Bengal, India)

No. 1141

DISCHARGE CERTIFICATE

Name of the Patient Sumita Pal
 Patient Registration Number 961/4-15 Date 22/04/14 Bed No. 2
 Age 35 Sex F Religion Hindu Nationality Indian
 Address Vill & PO - Nalikul P.S. - Haripal
Dist - Hooghly
 Admitted on 22/4/14 Time 9:55 am Discharge on 3/05/14 Time 1:10 pm
 Diagnosis Ampullary Carcinoma admitted @ ac pancreatitis
 Diagnosed by Histopathology/Other Histopathology proved
 Name of the Physician Dr. Ashim Kumar Chatterjee & Prof Anup Maymunda S. G. S.

Short History of the Case

PT presented conservatively
 di.
 Protonum Therapy
 with out
 Chemotherapy

Syr. Duphalac
 4 t t S.O.S

the above given diet

Advice on Discharge

Adv
 • Blent diet
 • Avoid spicy food.
 • Tab C Taxol XL (200) - 1 tab BD x 5d
 • Tab Pan D - 1 tab BD x 1 wks.
 • Syr Digesta MP - 3 t t BD x 3 wks.
 • Tab Sheleal (500) - 1 tab OD x 3 wks.
 • Cap Beron - 1 Cap OD x 4 wks.
 • Tab Pancreoflat - 1 tab BD with food x 2 months

• Repeat Serum amylase, Lipase, Calcium, Nat, K⁺ after 7 days - if any abnormality noted, consult general physician or inform sos.

Signature of MO/RMO

S. Croswami

Date :

Registration No. 633/6

Fax : 91-33-2534 2012



Signature of Consultant

Date :

Registration No. - 7445

E-mail : ccmrcc@gmail.com



Immunopath diagnostic centre

51/59, H. K. C. Dum Dum Road • Opposite Indira Maidan
Kolkata - 700 074 • Phone No. : 2548-3000
e-mail : immunopath1@gmail.com

REF. NO : D-4165 (OS)

DATE : 26-04-2016

NAME : Ms. Sumita Pal (OS)

SEX : F AGE : 37 YRS


Referred By : Dr. Ashim Chatterjee

B I O C H E M I S T R Y

TEST	TEST VALUE	REF. RANGE
SERUM UREA (KINETIC GLDH)	29.00 mg/dl	(10.00 - 45.00 mg/dl)
SERUM CREATININE (Jaffe's Alk. Picrate)	1.00 mg/dl	(0.70 - 1.30 mg/dl)
PLASMA GLUCOSE (FASTING) (GOD-POD)	290.00 mg/dl	(70.00 - 110.00 mg/dl)

Comments : RECHECKED.

N.B. : All reference ranges are age and sex matched. Reference limits mentioned herein are in accordance with the literature provided alongwith the kit which may change with the change in chemistry or the kit.


Dr. A. Mukherjee
MBBS, MD (Path)
Consultant Pathologist

Checked By IDC

Dr. A. Mukherjee
MBBS, MD (Path)
Consultant Pathologist

Dr. Adelene Basu
DCP, MD (Path)
Consultant Pathologist

Dr. S. Gupta
DCP, MD (Cal)
Consultant Pathologist

EKO DIAGNOSTICS

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Dept. of Health & Family Welfare, Govt. of West Bengal)



SUMITA PAL

36 YRS

29.04.2016

DR. OF MCH

1861/018

CT SCAN OF WHOLE ABDOMEN

HISTORY

? Periapillary Ca.

TECHNIQUE

Plain, oral and I.V. (non-ionic) contrast enhanced CT scan of whole abdomen done with 5 mm. and 10mm. sections in the axial plane.

FINDINGS

Digital radiograph of the abdomen in supine position and in frontal projection shows no obvious abnormality.

Liver is enlarged with focal dilatation of IHBR.

Gall bladder appears normal and its lumen does not show any radio-opaque calculus or intraluminal lesion (However, radio-lucent and small calculus may be missed in CT. USG may be done for further evaluation).

Common bile duct is dilated upto lower end.

Pancreas shows normal size, shape, attenuation characteristics and enhancement. No evidence of peripancreatic collection is seen.

Spleen is normal in size, shape and attenuation characteristics.

Both kidneys are normal in size, shape, attenuation characteristics and excretion of contrast media. Pelvicalyceal systems are not dilated. Perirenal fat planes appear normal.

Contd ...2..

1

AT Medical College & Hospital Campus
88, College Street, Kolkata - 700 073. Phone : 2212-3778/3779
Identity of the patient not verified. If there is any lack of correlation between the result and clinical condition, please refer the patient to the respective department.

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-2-

SUMITA PAL

36 YRS

29.04.2016

Ureters are not dilated.

Walls of fully distended urinary bladder are smooth and thin. There is no intraluminal abnormality. No evidence of growth from its wall. No evidence of vesical calculus. Perivesical fat planes are normal.

Pelvic organs are normal.

Irregular mass seen at lower end of CBD indenting C-loop.

IMPRESSION

CT features are suggestive of :

- 1) ? Periapillary Ca. (13mm x 13mm).
- 2) Hepatomegaly with focal dilatation of IHBR at places.

Suggested clinical correlation and further investigations if clinically indicated.

DR.D.SHARMA
MD

DR T.K.DHAR
MD (Radiodiagnosis)

DR.ANUP SADHU
DMRD, MD

2

AT Medical College & Hospital Campus
88, College Street, Kolkata - 700 073. Phone : 2212-3778/3779

Identity of the patient not verified. If there is any lack of correlation between the result and clinical condition, please refer the patient to the respective department.



Peerless Hospitex Hospital And Research Center Limited

360, Panchasayar, Kolkata - 700 094, Phone : 033-4011 1222,24622394/2462/0071-73

Fax : 033-2462 0692, E-mail : ph.enquiry@peerlesshospital.com • Website : www.peerlesshospital.com

CIN - U85110WB1989PLC046938



Print Date: 12-05-16 01:49:45 PM

DEPARTMENT OF GASTROENTEROLOGY

Ref No : EN/16/002520

Gender : Female

Req Dt : 12-MAY-16 10:40:08

Age : 36 Years

Name : Mrs. SUMITA PAUL

Visit ID: EX/16/022742 (MR/16/025545)

Order No. : IO/16/258567

Doctor : Dr. ASHIM CHATTERJEE

Report Dt: 12-MAY-16 11:08:28

Address : NALIKUL HARIPAL

Test Report

PYLORIC BALLOON DILATATION(TTS)

CLINICAL DETAILS	H/O CBD STENT FOR PERI AMPULARY TUMOR.
MEDICATION	INJECTION PROPOFOL.
ESOPHAGUS	NORMAL.
STOMACH	FOOD RESIDUE ++ FUNDUS & BODY - FOOD RESIDUE. ANTRUM - NORMAL.
DUODENUM	D1 - NORMAL D2 - NARROWING AT JUNCTION OF D1 & D2 JUNCTION WITH INFILTRATION. DILATATION DONE WITH 11 + 14 MM CRE BALLOON. AMPULLA COULD NOT BE IDENTIFIED.
DIAGNOSIS/COMMENT	D2 INFILTRATION WITH NARROWING - NEEDS SURGERY.

End of Report

Dr. J. R. MOHAPATRA
MBBS, MD, DM(GASTRO), MRCP(UK)
CONSULTANT

Prepared By: Suborna Sen Gupta

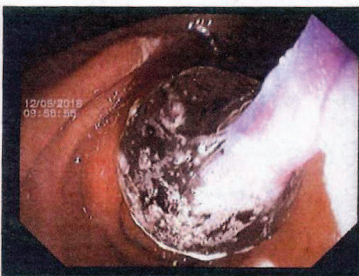
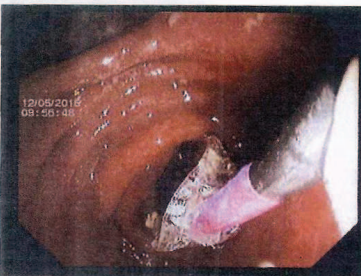
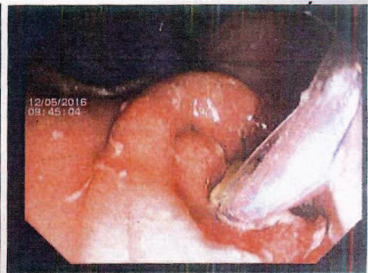
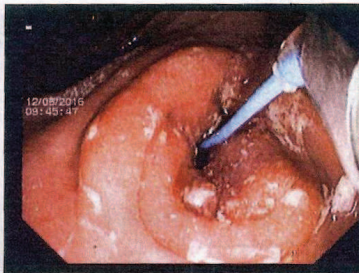
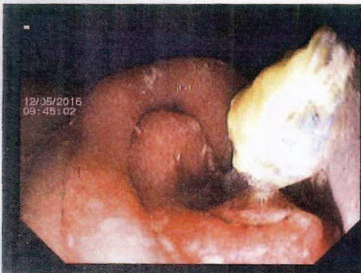
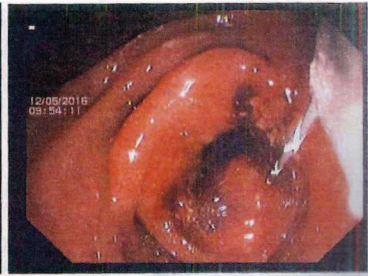
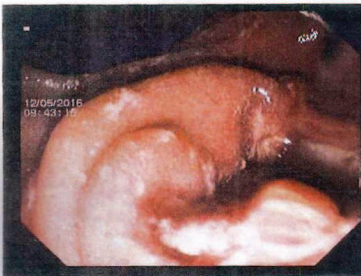
Page 1 of 1



PEERLESS HOSPITAL & B. K. ROY RESEARCH CENTRE
DEPARTMENT OF GASTROENTEROLOGY

Patient ID: 2016050027
Name: Mrs. sumita pal
Sex: F
Date: 12-May-2016

Ref By: Dr. J R M
Study: OESOPHAGEAL BALLOON DILATATION
Examined By: Dr. J R MAHAPATRA



Dr. J R MAHAPATRA

REGD. OFFICE :360, PANCHASAYAR, KOLKATA-700094, PH:- 033 2462-2394/2462
FAX : 033 2462-0766, Email : hospitex@vsnl.com, Website : www.peerlesshospital.com

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West Bengal Form

ATAK 107 1920K 1 K 2 2

Register No.

S. S. K. M. Hospital

KOLKATA - 700 020

ELECTRO THERAPEUTIC DEPARTMENT

Report / Treatment is required of

Name Sunanta pal Age 36 yrs Sex M

Address

Physician / Surgeon / Unit IV g/l

Ward Victoria

Bed No. / Cabin 53 Paying / Non-paying.

Brief History of Case : Perianapullain CA

Clinical Diagnosis : Hepatic Met/ Abscess

Particular point to be investigated : USG W/A focussing on A2

Instruction : hepatobilia Signature

Date 24/05/16

REPORT

Kindly give early date as patient planned
for upper bypass on 28/05/16
Thanks.

Blood & AFP
transferred

Btwe csk-16-0-155-478RRC
SSK-16-154-4,17 2.FFP
DEPARTMENT OF HEALTH AND FAMILY WELFARE
GOVERNMENT OF WEST BENGAL

DISCHARGE
B.S.K. ... P.G.R. & R
A.V.C. SUBS. ROAD, P.S. : SHOWANIPUR, KOLKATA-20

Sumita Pal
Victoria 53
Unit IV 9/6
Thursday OPD

Charge Certificate/Left Against Medical Advice		09/06/16		10am		Page No. : 1	
Charge No. :	Date of Discharge :		Time :	Patient Category : Free / Paying / Cabin			
Patient Name		Sex	Age	Yrs.	Months	Days	
Patient Srl. No.	Patient Registration No.	Admission Date		16-May-2016 Time: 10:34			
Address -	Post Office		NALIKUL				
Locality / Village	District		Hoochly				
Police Station	Religion :		Hindu				
Other	Nationality : Indian		Other				
Husband's Name	Husband's Name		AMAR PAL				
Phone/Unit	Phone/Mobile No.		9748814249				
Ward No.	Ward Name		Maternity (Ground Floor) (30)				
Diagnosis	Pancreatic carcinoma encasing the superior mesenteric vessels retroperitoneal lymphadenopathy, mass encasing SMA & SMV pedicle						
Referred To	Endocrinology OPD, Radiotherapy OPD, Chest Medicine OPD		Reason:				
Delivery Date & Time :	Mode Of Delivery : ND/ECL/LUCS/With Forceps/Without Forceps		In case of Confinement				
Delivery Status :	No. Of Child :		Antenatal Care Taken : Yes / No				
Delivery Date & Time :	Type of Surgery		Details of Baby				
Delivery Status :	Anesthesia Details		Birth Date : Birth Time :				
Investigation Done		Comments		Sex :			
SMA, SMV		Triple Bypass Procedure done		Birth Wt. :			
Xerox Done, multiple retroperitoneal LNs		Tejunopapernostomy done		Advice for Baby Patient			
Central Line		Gastrojejunostomy, Cholechojejunostomy, Jejunopapernostomy done		Patient discharged in a favorable condition			
Culture no growth		Proper Hemostasis Ensured		A/t Chest Medicine			
Syr Lactulose (25g) BD AC x 10 days		Abdomen closed in layers.		OPD for			
Restricted Diet, Diabetic Diet		Plenty of fluids		dysphagia & B/L with (pus) Endocrine OPD for DM			
Pantoprazole (40mg) BD AC		5 days		Baby Checked and Discharged			
Endonection MD (4mg) TAC AC		X 3 days		Rft Radiotherapy			
PCM + Aceclofenac (1 tab) BD AC		X 5 days		Signature			
Cefuroxime (500mg) BD AC		X 5 days		Date			
Insulin (R) (4) (4) (2) S/C		cent		Time : after collecting			
Insulin Glargine (10 U S/C at 10pm)		cent		Prognosis report			
Counter Signature of the Visiting Staff		Review in 1-OPD		Signature of the Medical Officer			
		On 09/06/16 &/or (Thurs day)		Dependent			
		ER SOS.					

Case No. - 6

So far case study produced in this publication is related to solid tumours. But blood cancer (leukaemia) is one of the varieties which do not belong to this group. In early part of 1994 study and application under Prof. Manju Dutta Choudhury, Head of the Department of Haematology in the School of Tropical Medicine was conducted with amazing success. But due to some legal complications that process could not continue excepting few isolated cases. Now in the month of March, 2012 an elderly person named Khalil Ahamed Molla, aged 75 years having a case of Acute Myeloid Leukaemia (M4) being refused from R. G. Kar Medical College Hospital, Kolkata and Tata Medical Centre, New Town, Kolkata, approached helplessly to this Critical Cancer Management Centre & Clinic. With application of Psorinum Therapy at this centre this elderly person gradually recovered to the normal life and his present diagnostic reports show satisfactory results. The details I would like to provide below –

Khalil Ahamed Molla, residing at Dhakshin Bamonia, Paglarhat Kashipur, 24 – Pargana (South), West Bengal was suffering from persisting high fever and bleeding continuously from nose and teeth. Huge blood loss led to low Haemoglobin percentage together with falling white blood corpuscles. The elderly person visited R. G. Kar Medical College Hospital on 24.03.2012. CT Scan of Thorax and Bone Marrow examination was done on 28.03.2012 serial no. BM/ 58/ 12 the report revealed Acute Myeloid Leukaemia (M4) and the prognosis was grim. On 03.04.2012 the patient was registered in Tata Medical Centre, Newtown, Kolkata MR/ 12/ 02459 – patient no. OP 12/ 004904 prognosis continued to be grim and patient evaluation summary diagnosis mentioned as Acute Myeloid Leukaemia. Only supportive care was given and antibiotics applied along with blood transfusion of 15 units but gradual deterioration could not be arrested. He was admitted to our clinic on 10.04.2012 and Psorinum Therapy was applied. Almost four years have elapsed and the patient was found improving satisfactorily. Unfortunately the patient died on 12.07.2013 suffering from diarrhea.

Reports and supporting documents of Khalil Ahamed Molla

Name : KHALIL AHMED MOLLA
Lab No : 12 8308623
Age : 77 Year M
Referred By : DR.R.G.KAR
Report at : TREATMENT DIAGNOSTICS

Cytopath No. : 11280A-81A/2012
Date of Registration : 22/03/2012
Date of Receipt : 23/03/2012
Date of Reporting : 24/03/2012

CYTOPATHOLOGY REPORT

SPECIMEN : Pleural fluid for cytology.

CLINICAL HISTORY : --

GROSS : Received 2.5 ml pale yellow fluid, mixed with equal volumes of fixative.



MICROSCOPIC : Cytospin smears are cellular variably preserved and show presence of polymorphs lymphocytes and histiocytes and mesothelial cells. No malignant cells are seen in smears examined.

IMPRESSION : No malignant cells are seen in smears examined.

DR. MANI MAKHLJA
MD(DNS)(PATH)
CONSULTANT(PATH)

DR. GAURAV SHARMA
MD(PATH)
CONSULTANT(PATH)

DR. SACHIN JOHARI
MD(PATH)
CONSULTANT(PATH)

DR. RAMAN ARORA
MD(PATH)
CONSULTANT(PATH)

DR. ALOK SHARMA
MD(PATH)
SR. CONSULTANT(PATH)

DR. MOUSHUMI SURYAVANSHI
MD(PATH)
SR. CONSULTANT(PATH)

DR. PATIKA S. AGRAWAL
MD(PATH/POCC/RENAL PATH)
SR. CONSULTANT(PATH)

DR. HEMA MALINI AYER
MD(PATH)
HOD-HISTOPATH/CYTOPATH

DR. S.B. KULKARNI
MD(PATH)
CONSULTANT(PATH)

DR. (WG. CDR.) Y.M. SIRPAL
MD(PATH & MICRO), DNS.M.Sc. FRCMS
CHIEF CONSULTANT-PATH & MICRO

DR. VANDANA LAL
MD(PATH)
CHIEF OF PATHOLOGY

Note : - Slide / Block can be issued only on advice of the referring consultant after a minimum of 48 hours.

1/1

Date 28/3/12

Mollen

68 yrs.

Ward M M W 6

Bed No. F-15

Unit, Referring Physician

1800/cmm

49

23

0.5

5

○

Blast 25%

Adequate

Normocytic Normochromic

Cellularity - Normo cellular

Grossly depressed but normoklastic on maturation.

Predominant population of myeloblast $>46\%$

Some blants are showing scanty vegetation

with 2-4 nuclei &
presence of granules

the other rods in cytoplasm while some other almost are showing abundant

cytoplasm with convoluted
nuclei

Acute Myeloid Leukaemia (M4)

Immunophenotyping suggested for further confirmation

W



NORTH CITY



DEPARTMENT OF LABORATORY MEDICINE

Patient ID: 32503030

Patient Name: KHALIL AHMED MOLLA Age: 68 Years. Sex: Male.

Refd. By : Dr. R.G. KAR (U-I) MEDICINE(MW)

Sample Drawn Date: 29/03/2012 Sample Collection Time : 01:00:54 PM

Reporting Date: 29/03/2012 Reporting Time : 16:22:32

TEST REPORT DEPARTMENT OF HAEMATOLOGY

TEST	RESULT	BIOLOGICAL REFERENCE INTERVAL
H+EMGLOBIN	9.6	12.5-15 g/dl
TOTAL LEUCOCYTE COUNT	1500	4000-11000/cu mm
<u>DIFFERENTIAL LEUCOCYTE COUNT</u>		
NEUTROPHIL	31	40-70%
EOSINOPHIL	06	1-6%
BASOPHIL	00	0-2%
LYMPHOCYTE	61	20-40%
MONOCYTE	02	2-10 %
ESR (1 ST HOUR) (WESTERGREN METHOD)	75	0-20 mm

PERIPHERAL SMEAR EXAMINATION

Erythrocytes are predominantly normocytic and normochromic.

Few atypical cells seen. Platelets are adequate.



Dr. MIR HASSAN
DMRD, M.D. (PATH) GOLD MEDALIST
CONSULTANT PATHOLOGIST

Page 1 of 1

DEPARTMENT OF HEALTH AND FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
DISCHARGE

N. S. KAR MEDICAL COLLEGE & HOSPITAL
1, KUNDIRAN BOSE SARANI, P.S. 1, KOLKATA-84

Unit - I (M)
MMW
B/NO - F15
OPD - Monday

Referral

Discharge Certificate/Left Against Medical Advice

Page No. : 1

Discharge No. : *Khuli/* Date of Discharge : *31.3.12* Time : *11 AM* Retention Category : Free / Paying / Cabin
Patient Name : *KHANTI AHARE KOLLA* Sex : Male Age : 68 Yrs. Months : Days : Hrs.
Patient Site No. : Patient Registration No. : Admission Date :
Address : *PAT 2607971* Post Office : *6612133478* Telephone : *19-KXP-2012 T1067 19120*
Municipality / Village : District : *D-CHALTAHERIA*
Police Station : *DAKSHIN BANURIA* Nationality : *Indian* Religion : *South 24 Parganas*
State : *KASIPUR P. S.* Husband's Name : *Muslim* Other :
Father's Name : *West Bengal* Phone/Mobile No. :
Doctor/Unit : *LATE EMAN ALL KOLLA* Ward Name :
Bed No. : *18008004291* Bed Type :
Final Diagnosis : *AML (M4)* MLE MEDICAL BOARD (PMW-6) (IPW60)

Referred To : *Hematology OPD of Medical College Calcutta* Referred Out Case

Delivery Date & Time : Mode Of Delivery : *ND/ECL/UCS/With Forceps/Without Forceps*
Delivery Status : No. Of Child : Antenatal Care Taken : Yes / No

In case of Surgery
Surgery Date & Time : *CT Thorax - Minimal chydro*
Surgery Status : *pneumothorax of right & collapse*
Consolidation of at lower lobe.

Details of Baby
Birth Date : Birth Time :
Disc No. : Sex :
Birth Wt. :
Advice for Baby : *NEG (100 malignancy cell.)*

Anaesthesia Details
Pleural fluid - PHP Smear - NEG (100 malignancy cell.)
cell - 4200, N 20, 80, Glu - 65, pvt - 4.07.
CT - 106, ADA - 9.

Advice for Baby
RBC - 115, WBC - 37, ur - 11
Na⁺ - 137, K⁺ - 4.7
Hb - 9.6, TLC - 1500
NO/LB/E6M2, ESR - 75
T. Bili - 0.7, T. ptn - 6.8
C. 1 - 0.3, ALB - 3.5
ALT - 38, AST - 39, ALP - 551
GAT - 256, Uric - 9.8
LDH - 558

Investigation Done
Test Name : *Bone marrow biopsy -*
AML (M4)

Baby Checked and Discharged :
Signature :
Date : Time :

Medicine Name Adv. No. of Days Comments
1 Normal diet
1 Cap. parido 650 - 1 x BBT x 1m.
1 Tab. Sensiclav (625) - 1x TDS x 7d.

ADVICE
X Tab. Stilef-02 - 1x BBT x 5 days.
X Metrogyl DS gel - To apply in
mouth QDS x 15 days.
1 Heridine mouth wash - To single
1 water (1:1000) x 15 days.

Counter Signature of the Visiting Staff : *Dr. Rup K. Das - 30 MHS x 15d.* Signature of the Medical Officer

To attend Hematology
OPD of Medical College
Calcutta for further
management.



Tata Medical Center

14 MAR (EW) , Newtown, Kolkata - 700 156
Phone: +91 33 6605 7000, 7222 , Email : info@tmckolkata.com
Website: www.tmckolkata.com

Patient Evaluation Summary

Run Date : 03/04/2012 16:43:43

MR No. : MR/12/002459 Name : KHALIL AHAMED MOLLA
Age : 75 Y O M O D Sex : MALE Visit Date : 03/04/2012
Patient No. : OP/12/004904 Address : VILL-DHASKHN BAGONIA PAGLARHAT KASHIPUR, 24 PARGANAS
SOUTH, WEST BENGAL, INDIA

Assessment Date : 03/04/2012 16:43:26

VITAL PARAMETERS (Last Measured)

Height: 159 cms Weight: 45.4 kg BSA: 1.42 sq m BMI: 17.96 kg/sq m
Pulse: 86 / min Temperature: 97 Deg F BP: 115 / 55 mmHg Respiratory Rate: 20 / min

CHIEF COMPLAINTS

75yr old elderly man recently diagnosed as ?Acute Leukemia has come for second opinion/ further management.
C/o Weakness, Dry Cough with chest pain along left side of chest wall

Diagnosis

Diagnosis

Acute myeloblastic leukaemia
(Differential)

Morphology

Remarks

? Acute Leukemia in Elderly man
?AML

HISTORY

Feb 2012 onwards-
Weakness
Left sided chest pain, Dry cough
Fever-On/ off
Consulted local physician-given symptomatic treatment
No relief in symptoms

Mar 18, 2012: visited RG Kar Medical College, kolkata
Evaluated

CBC: TLC: 1500/cu.mm, Hb:9.6g/dl, Platelet: not mentioned
Urea:37mg/dl, Creatinine:1.1mg/dl, RBS:115mg/dl, ALT:36iu/l, ast:39iu/l, SAP:551U, LDH:558,
CECT-Chest: minimal HYDROPNEUMOTHORAX (L), Collapse consolidation left lung-lower lobe
BMA: Normocellular marrow, Predominant population of Myeloblasts>46%, some blasts show scanty cytoplasm
with 2-4 nucleoli, granules, aeur rods.

AML-M4. Slides not furnished

Received Supportive care and antibiotics Amox-clav, Metronidazole

No known medical co-morbidities
No past h/o hospitalization
Has 13 children, 4 brother, 2 sisters. Stays with son
Monthly income around Rs. 3000/month

ON EXAMINATION

ECOG-1
Afebrile
Pallor+, no icterus, petechiae,
senile purpura seen
No peripheral palpable lymph nodes
ABD: Soft, non tender, Liver/ Spleen-not palpable, no testicular enlargement
RS: B/L Vesicular BS, DECREASED BS in left lower areas, no adv sounds
CNS: NoFND
CVS: S1 S2+, JVP-Normal

Please take an appointment for next visit. Ph : 033-6605 7222, Email Id : appointment@tmckolkata.com

Page 1 of 2

✓

INVESTIGATION

Investigation

BLOOD GROUPING AND RH TYPING
BLOOD BORNE VIRUS SCREEN
CALCIUM AND PHOSPHATE
ELEC/RENAL
LDH
LFT
URIC ACID
APTT
CBC PROFILE
PT WITH INR
CHEST PA

Remarks

DISCUSSION

Reports furnished are suggestive of Acute Myeloid Leukemia in this elderly man aged 75yrs. He needs diagnostic evaluation [also because he has not furnished any of the slides for review] and further management. The adverse prognosis of the disease (if it is indeed AML), the costs involved, nature of supportive required and palliative intent of Rx required for him was explained in detail to the patient's son and relative.

Plan: BMA/Bx, IPT (+/- CYTOGENETICS)

palliative therapy: disease control + supportive care

Cap. Hydroxycarbamide / other options (Len/ Decitabine/ SC Cytosar could be explored based on affordability)

ADVICE

They would like to discuss in the family and return with a final decision for diagnostic evaluation and palliative therapy for disease control. They do not want intensive treatment considering his age and financial logistics.

Medications:

1. Tab. Levofloxacin 500mg PO, OD, Daily
2. Syp. Bricarex 5cc PO /PRN
3. Tab. Pen 40mg PO, OD, AC

Hexidine mouth wash, Syp-Dyphalac 10cc HS

REVIEW SOS / PRN



Dr. Vivek S Radhakrishnan, MD MSc
DM ECMO
, Regn. No. : 71523
Department of Clinical Hematology

-End of Report-

Case no.: 7

Smt. Sishu Bala Ghosh, a 55 year old lady residing at Bidhan Colony, Patipukur, Kolkata-700048, was suffering from Anaplastic carcinoma involving the mandible (Rt.) region since last six months then. Punch biopsy done at Nil Ratan Sarkar Hospital, Kolkata. Patient's ticket no. 49226 slide no. 141/91. X-ray report of Sishu Bala indicated resorption of bone horizontal ramus of right mandible at the third molar region and altered Trabecular pattern of the bone at the root to the second molar fourth. Biopsy report stated Mrs. Ghosh's carcinoma is of Anaplastic nature, presented with swelling of Rt. lower jaw with multiple discharging sieves. Patient was administered Psorinum therapy from 25.06.1991. Gradually patient recovered from the severe pain and showed regression on the affected zone. Finally the patient was completely cured and led a normal life for a long time.

Reports and supporting documents of Smt. Sishu Bala Ghosh

SMT. SISHUBALA GHOSH
Anaplastic Carcinoma,
Right mandible.

Smt. Sishu Bala Ghosh, 55 years old lady at present residing at Bidhan Colony, Patipukur, Calcutta - 700 048 was suffering from Anaplastic carcinoma involving the mandible (Rt.) region since last 6 months. Punch biopsy done by Nilratan Sircar Medical College Hospital, Calcutta. patient's ticket no. 49226 slide no. 141/91.

. First treatment commenced with Ampicilin 500 / 7 Ds x 7 days but no result. Patient was examined by Dr. Debashis Santra, B.D.S., Calcutta Ex. Dental Surgeon of Dr. R. Ahmed Dental College & Hospital and Nilratan Sircar Medical College Hospital, Calcutta, B. C. Roy Children Hospital, Calcutta. Dr. Santra suggested Cancer and the patient was referred to N.R.S. Hospital and biopsy done by them, X-ray & blood examination was also done. Hb% was 8.3 and X-ray report said that resorption of bone of horizontal ramus of right mandible at the 3rd. molar region and altered Trabecular pattern of the bone at the root 2nd. molar fourth. Biopsy says Mrs. Ghosh's carcinoma is of Anaplastic nature, presented with swelling of Rt. lower jaw with multiple discharging sieves. Dr. Asim Kumar Chatterjee applied his medicine on 25.06.91. Within three days Smt. Ghosh got relief from pain, all the three sieves seen drying up. After one month lesions have fully dried up & outer lesions have been filled up. She was able to take hard foods without any trouble.

Contd....

She was advised for operation of affected part of the jaw with a view that the case may fully be cured. but patient, due to financial embarcement, not desired to go under operation and she discontinued the treatment.

Asim Kumar Chatterjee

Dr. ASIM KUMAR CHATTERJEE
D.M. CAL,
PRESIDENT, THE NEW RESOURCE

<p><i>Dr. Gobindus Pantia. B. D. S. Cal.</i></p> <p>DENTAL SURGEON SPECIALIST: ORAL DISEASES ORAL SURGERY</p> <p>ARTIFICIAL TEETH</p> <p>EX SURGEON Dr. R. Ahmed Dental College & Hospital Ramkrishna Mahamangal Sova Pratishthan B. C. Ray Children Hospital Nilratan Sirkar Medical College & Hospital</p>	<p>Dental Clinic :</p> <p>46, SURJA SEN STREET CALCUTTA-700 009</p> <p>6 to 8 P. M. Phone : 35-0681</p> <p>Eastern Poly Clinic 95, BELIAGHATA MAIN ROAD, CALCUTTA-700 010</p> <p>9 to 11 A. M.</p>	<p>Residence :</p> <p>Manju Villa 35-5, RADHA MADHAB DUTTA GARDEN LANE CALCUTTA-700 010</p> <p>7 to 8 A. M. by appointment Phone : 35-8427</p> <p>OPPOSITE TO V.I.P. GATE OF SALT LAKE STADIUM.</p> <p>Date..... <i>12/6/91</i></p>
<p><i>Lesher Bala</i> <i>Jy. T. Tox</i></p> <p><i>C. A</i> <i>Ep. Streptococcus - 12</i></p> <p><i>Dr. K. Chatterjee</i> <i>Kap. 180</i></p> <p><i>Cancer Hospital</i> <i>Tab. Bimphex 12</i></p> <p><i>Tab. Poly</i> <i>Tab. 12</i></p> <p><i>Tab. 12</i> <i>Tab. 12</i></p> <p><i>Tab. 12</i> <i>Tab. 12</i></p>		

West Bengal Form 700. 49221

TICKET FOR OUT-DOOR PATIENTS

At the..... Hospital

Date of first visit..... No. in O. P. Register.....

Name.....

Age..... Sex.....

Place.....

Date	Treatment
4	1. Cup. max. 100
	1 cup 70 x 5 mm
	2 cup. Brinnet 100
	1 cup. Nuly 100
	X 15 mm

ACJP 2-A 30008-1977-78-2,64,000-2,64,00,000

29/2/91

Lunch Biopsy 2 cup

R

0.500 1 cup Max (also)

0.000 1 cup TOS

0.000 1 tab Aristozyl (250)

0.000 1 tab TOS

0.000 1 tab Nupenal - (10)

0.000 1 tab 100

0.000 1 tab Becozyme in 100

0.000 1 tab Rainy (10)

West Bengal Form No. 769. 19226
 TICKET FOR OUT-DOOR PATIENTS
 At the..... Hospital
 Date of first visit..... No. in O. P. Register.....
 Name.....
 Age..... Sex.....
 Disease.....

Ad to come after
 1 day for body
 weight 12/1/21

Date 12/1/21 Treatment
 Much high fever
 for 2 to 3 days

Re 1) Day 12/1/21 - 15
 on 12/1/21
 2) Talit was on 12/1/21
 ACJP-A 00005-1077-78-2,64,000-2,64,000,000
 12/1/21

Amrinda 49226
 West Bengal Form No. 769.
 TICKET FOR OUT-DOOR PATIENTS
 At the..... Hospital
 Date of first visit..... No. in O. P. Register.....
 Name.....
 Age..... Sex.....
 Disease.....

Overall histopathological
 features
 is suggestive
 of "Anaplastic
 Squamous cell
 carcinoma."

Date..... Treatment
 ingested growth
 and in the
 abdominal region
 for last 6
 months

Am
 21/5/21

24. 6. 91

last 1 1/2 months
P/P - sq. cell ca (?)
Referred to 4A for HP
evaluation

An endosteal sleeve
lesion involving the
right side of the mandible
buccal forame is deepened
and deformed. Extent not

B.C.L. = CA 700 015
unplanned

P/P Sq. cell carcinoma

Ad

- 1) 30° lateral oblique view
Right side of the mandible
- 2) Exam of blood for T.C.,
D.C., B.S.R., Hb%, Blood sugar
(fasting)
- 3) ~~Cap~~ Cap max. 25 - 15 cap
one cap 1/2 inch x 1/2 inch
- 4) Cap divided into 21 cap
at cap end of neck

AP 2

Dr. to univ. 24/7/91 5/6/91
at a complete clinical history
which should include a full medical
history to know if it is a primary
malignant neoplasm.

R. 1. Discharge 1 Ph
at 1st visit in R. 2. 6/11

TICKET FOR OUT-DOOR PATIENTS

At the Nilratan Sirkar Medical College Hospital

Date of first visit..... No. in O. P. Register.....

Name.....

Age..... Caste.....

Disease.....

Date..... Treatment.....

An indurated massive ulcerated area in the alveolar ridge of lower right mandible for the last 1½ months
P/P - Sq. cell ca (?)
Referred to 4A for HP evaluation

An indurated plaque lesion involving the right side of the mandible buccal tissue is depapilled and deformed. Exposed

B.C.L. (17070) (17070) (17070) (17070) (17070)

Unpl. adenocarcinoma

P/P - Sq. cell carcinoma

Adv

- 1) 30° lateral oblique view of Right side of the mandible
- 2) Examination of blood for T.C., D.C., ESR, Hb%, Blood sugar (fasting)

R₂ ~~cap~~ max. 25 - 15 cap
one cap of 15 x 15 cm

- 1) cap divided into 20 x 20 cm
at cap of 15 x 15 cm

A-2

Dr. N. S. Sircar, 20/7/1956/1/1



28-6-91



22-9-91



4-8-91

Case no.: 8

A 17-years-old sweet, beautiful girl was admitted in a nursing home in Kolkata on 21.03.1997 after complaining of severe lower abdominal pain, nausea, vomiting and fever. Her ultra-sonography report suggested right-ovarian tumour (Terrato Dermoid). She was operated and her operative findings were: Right-sided ovarian tumour partly-cystic partly-solid (Dermoid & Hair) capsule intact. Left-ovary absolutely normal, both tubes of uterus NAD appendix appeared pathological right-sided oothorectomy (removal of ovarian tumour) and appendisectomy done.

On 23.03.1997 the material sent for histopathological investigation on were: (1) Right-sided ovarian tumour, (2) Tissue from right-fallopian tube and (3) Adherent haemorrhagic omentum.

The microscopical examination suggested:

- Multiple sections examined from different parts of the ovarian tumour. The tumour was composed of ectodermal, mesodermal and entodermal elements. Skin with its appendages, cartilage, bone, fibrofatty tissue, glial tissue, mucin secreting glands, respiratory epithelium etc. were present. Majority of the elements were mature. A small focal area of blastemal element was seen. Areas of haemorrhage and necrosis were present.
- The tissue labelled as fallopian tube showed ovarian tissue with primordial follicles and small follicle cysts.
- The omental tissue showed congested fibrofatty tissue showing focal collections of chronic inflammatory cells. Dilated blood vessels were present. No tumour tissue found.

The diagnosis was : Teratoma of ovary. Majoriy of the elements present are mature. A small focal area showing undifferentiated blastemal elements was present.

The patient was discharged from the nursing home on 29.03.1997. The slides sent for examination on 4.04.1997 indicated:

- Sections showed histology of a malignant teratoma (immature teratoma).
- Sections through omental tissue showed marked congestion. No neoplastic deposits seen.
- Sections through appendix showed lymphoid hyperplasia. Sections through other ovary show no neoplastic infiltration.

Unfortunately after a few months the patient again fell severely ill and was taken to Tata Memorial Hospital, Bombay and was admitted there on 29.07.1997 following severe lower abdominal pain and vomiting. She was admitted under the supervision of Dr. R. K. Deshpande there. Dr. Deshpande observed reoccurrence of immature teratoma ovary and the patient underwent another surgical debulking (Panhysterectomy & Omentectomy).

At this time Dr. Deshpande referred the patient to Dr. Tapan K. Saikia writing to him:

“09.08.1997

Tapan,

This was a bad disease stuck to wall. I am not sure of Microscopic clearance. Very likely R-1.

Kindly reconsider.”

Dr. Saikia advised 3 cycles of chemotherapy to the patient. But due to the patient's failing health chemotherapy could not be administered.

The patient belonged to a rich and influential family in Kolkata. She was the only child to her parents. Finally the patient's family approached me for help. 19 years ago our work was not as prominent as now is. This particular case was full of complexities. Initially everyone including Prof. Anup Majumdar advised me not to take this case. But after unending requests coming from the patient's family and on humanitarian grounds I took up the responsibility of the treatment of the teenage girl. Later on Prof. Majumdar also joined me in this case. We immediately started our treatment with Psorinum Therapy. For the initial six months under our treatment the patient's condition was critical. Prof. Majumdar and I together worked very hard on this case. Later her overall condition started improving and eventually she started feeling better. Within two years she was fully cured. She then appeared for her class-12 board examinations and later on completed her graduation with History (Honours). She is quite well now and is working. Hopefully she continues to be so.

Unfortunately Prof. Majumdar's prescriptions, the patient's follow up history and medical notes are lost. Dr. Saroj Gupta used to say that the recurrence of immature teratoma has a very bad prognosis. He and Prof. Subir Dutta were both very interested in this case and at the same time were very encouraging. I am providing here limited documentations of this case keeping in mind that the patient is a young girl and has a social life. If in future anyone gets interested to study on undifferentiated Malignant Teratoma, he or she would find this case-history to be very helpful.

Reports and supporting documents of Miss Pouloma Mukherjee

Dr. Subir Kumar Dutta
MBBS, DCP, MD. (Path)
Dr. Sunil Kumar Gupta
MBBS, DCP, MD, FRCPath. (Eng)

Scientific Clinical Research Laboratory Pvt. Ltd.
2, RAM CHANDRA DAS ROW
(OFF 77, DHARMATALA STREET)
CALCUTTA-700013
Phone : 244-1088

Date 4. 4. 19 97

REPORT ON THE EXAMINATION OF
Lesion in (Rt.) ovarian tumour

Patient's Name Miss [redacted] Mukherjee

Referred by Dr. Saroj Gupta

Slides : T and R 4101 - 12 slides.

REVIEW REPORT :

All sections were reviewed.

Sections show histology of a malignant teratoma.
(immature teratoma).

Sections through omental tissues show marked congestion.
No neoplastic deposits seen.

Sections through appendix show lymphoid hyperplasia.

Sections through other ovary show no neoplastic infiltration.

(SUNIL KUMAR GUPTA)

(SUBIR KUMAR DUTTA)

Stained Slide/s & Paraffin block/s enclosed.

The remaining tissue (if any) will be preserved for
a period of ten days from the date of reporting

Malabika Datta

M.B.B.S. (Cal.) M.R.C.P. (UK) D.M.R.D. (LOND.)
Consultant Radiologist & Sonologist

GEMINI'S IMAGING CLINIC

131A, RASHBENARI AVENUE

SUPARNO

CALCUTTA-700 029

PHONE : 464-1737 □ 464-3216

NAME Miss. [REDACTED] Mukherjee.

AGE 17 yrs.

REFERRED BY Dr. Goutam mukhopadhyay.

REF. NO. U - 1225.

PART EXAMINED Whole Abdomen.

DATE 26.7.97.

ULTRASOUND REPORT

1. Fluid present in abdomen.

LIVER:

is not enlarged. Shows smooth outline and homogeneous echo pattern. Intrahepatic ducts are not dilated. There is no solid or cystic mass to suggest presence of cyst, abscess, primary or secondary neoplasm.

GALL BLADDER:

neither dilated nor contracted. Shows normal smooth thin outline and cystic nature. There is no thickened mucosa, stone or mass.

COMMON BILE DUCT:

not dilated. No stone identified. Calibre measures 5mm.

PANCREAS:

is not enlarged. Shows normal well defined outline and homogeneous echo pattern. No dilated ducts or calcifications identified. No solid or cystic mass demonstrated to suggest presence of pseudocyst, abscess or neoplasm.

GUT:

not enlarged. Shows normal echo pattern.

THORACIC- AORTIC LYMPH NODES:

are not enlarged.

KIDNEYS:

Both kidney show normal shape, size, smooth outline and cortical thickness. No evidence of atherosclerosis, nephrocalcinosis, chronic pyelonephritis, calculus, hydronephrosis, cystic disease or neoplasm demonstrated. Cortical and medullary echo pattern is well differentiated.

UTERUS:

are not dilated hence not traceable.

VAGINA:

is not enlarged. Shows normal homogeneous echo pattern of myometrium with smooth outline. Endometrium lining the linear midline echo of empty cavity is not thickened. No focal mass demonstrated to suggest presence of myoma.

OVARY :

Left ovary measures 26mm X 26mm in size, shows normal echo pattern and contains few small follicles.

Right ovary is removed but a fair size mass demonstrated over right side of pelvis showing solid and cystic component. Approx size measures 8.6cm X 6cm. Margin of the mass is not very smooth or sharp.

SPACE OF DOUGLAS:

Fluid or mass present.

IMPRESSION:

Local recurrence of right ovarian Neoplasm.

M. Datta

TATA MEMORIAL HOSPITAL CLINICAL HISTORY

DATE 29/3/97

Clinical notes at first visit by Dr. Ans

SYMPTOMS :

DURATION

- Since mid 1994, nausea off and on
_____ D M Y
_____ D M Y
_____ D M Y
_____ D M Y
- in early 1997, symptoms ↑ - fever
- loss of appetite

PREVIOUS TREATMENT :

☐ NIL ☐ BIOPSY ☐ ENDOSCOPY
☐ SURGERY ☐ RADIOTHERAPY ☒ CHEMOTHERAPY

DETAILS :

H/O @ ovarian cystectomy : 2 Teratoma
23rd March 1997 Received Chemo. mostly diff. type
BEP x3 cycles
? exact dose
focus of small lymphoma
Now CT-scan/Pelvis
Recurrent Nausea @ ovarian
Nerves - (N)

HABITS : ☒ Nil

☐ Cigarette ☐ Beedi smoking ☐ Alcohol ☐ Betel leaves ☐ Tobacco chewing ☐ Betel nut

FAMILY HISTORY : ☒ NIL

Relationship	Disease
a. _____	_____
b. _____	_____

MEDICAL HISTORY : ☒ NIL

DISEASE	DURATION	TREATMENT		
		nil	irregular	regular
Diabetes	_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hypertension	_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Heart disease	_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
TB	_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Asthma	_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Dr Tapen Sarkis M - (29)

29/7/97

minimally symptomatic

↓
Occ lower abdominal pain

CT scan of abd. 28/7/97 -
USA

(R) Adrenal mass (cystic) recurrence.

- Seven Tumor Markers -

β HCG, AFP and CA-125 -

↓
Normal 4/97

↓
Normal 4/97

↓ Normal
(marginally elev.)
↑ 2.4 u/ml (4/97)

Needs re-exploration and excision of the mass in toto.

4/9/97 Has undergone Parathyroidectomy.
Recovered well.

Final Histopath: Immature
Teratoma

Ryplair

May return on Satm

Rs. 4h

Plan: Chemo + RTH

Ryplair

Dr Tapen Sarkis for Opinion

1/11/97 Re section - we
would like to see further observation and

TATA MEMORIAL HOSPITAL

Form No.

NAME _____

CASE NO. _____

Date _____

4

Physician's Follow up Notes

Tapan,

7/8/95

This was a bad disease stroke to wall.
I am not sure of Microscope clearance.
Very likely R-1.

Kindly reconsider

7/8/92

- Discussed

Rama

- Chemotherapy for residual disease

- RT also if required - Radiotherapist to advise

R.

st.

Mr. Edwards - 140 mg/day IV infusion in Myles 14 x 5 days (1-5)

Dr. Wilson - 1.7 g/day IV infusion in AWS 14 x 5 days (1-5)

Dr. Hemo - 400 mg IV bolus at 10:00, 4 x 2.5 daily 1-5 days (1-5)

(Rama C. and A. Hemo)

Mr. Clapham - 30 mg/day IV infusion x 5 days (1-5) along with

IV fluids, morphine and antibiotics

1/8/95

RT - 4 weeks

18/11/95

sections are submitted.

Also received separately sent omental tissue which is unremarkable. Also

TATA MEMORIAL HOSPITAL (TATA MEMORIAL CENTRE)

Phone : 414 6750 (6 lines)
Telex : 011-73649 TMC IN
Fax : 022-4146937



DR. ERNEST BORGES ROAD,
PAREL, BOMBAY 400012.

Ref: M. 1466

7/8/97

ADVICE FOR CHEMOTHERAPY

Miss [redacted] Mukherjee, 17 year old who experienced recurrence of immature teratoma ovary following right ovarian cystectomy and had a 3 cycles of chemotherapy and undergone another surgical debulking (Pain, omentectomy + omentectomy) recently here (Surgeon: Dr. R.K. Deshpande). The recurrence was at microscopic disease possibly had remained. Therefore, she is advised to take 3 cycles of chemotherapy with -

Cycle I

Inj. Doxorubicin	140mg/day IV infusion in 4/saline 1 hr for 5 days (1-5)
Inj. Cyclophosphamide	1.7 g/day IV infusion in 2/s 1 hr for 5 days (1-5)
Inj. Etoposide	430mg IV bolus at hours 0, 4 and 8 daily for 5 days (1-5) (hour 0 is start of Doxorubicin)
Inj. Leucovorin	30mg/day IV infusion for 5 days (1-5) along with IV fluids, mannitol and antiemetics.

The cycle is to be repeated at monthly interval.

Separate sheets is attached.

Incidentally, hcg serum tumor markers, Beta HCG, AFP and CA 125 are within normal limit. Radiotherapist should evaluate regarding need for RT.

(Dr. T.K. Sarkis MD)
Medical Oncologist



Advanced Medicare & Research Institute

A Joint Sector with Government of West Bengal

DEPARTMENT OF PATHOLOGY

Patient's Name: XXXXXXXXXX MUKHERJEE Age: 17 Y Sex: F
Referred By Doctor: A. MAJUMDAR
Date of Receipt Sample: 13/10/97 Date of Report Delivery: 15/10/97

URINE REPORT

TEST..NAME	RESULT
PHYSICAL	
Volume (Receive6)	30 ml.
Colour	Pale yellow
Reaction (pH)	pH 5.5 acidic
Sp.Gravity	1015
CHEMICAL	
Protein	Nil
Sugar	Nil
Ketones	Nil
Bile Pigment	Nil
Bile Salts	Nil
Urobilinogen	Normal
MICROSCOPY	
W.B.C.	Occasional
R.B.C.	Nil
Epithelial cell	0-3/hpf
Casts	Nil
Crystals	Nil

DR (MAJ GEN) S.R. BHATTACHARYA
MBBS, MD, DCP
HEAD & SENIOR CONSULTANT (PATHOLOGY)

DR. (PROF) B. D. CHATTERJEE
MBBS, DSc, DipBact, FAMS
CONSULTANT MICROBIOLOGIST

DR. (COL) A. PANDIT
MBBS, MD, DCP.
CONSULTANT PATHOLOGIST

DR. ENAM MURSHED KHAN
MD (PGI), Dip NB (N.Delhi),
FRC Path (LONDON),
CONSULTANT HISTOPATHOLOGIST
& CYTOPATHOLOGIST

Registered office : Advanced Medicare & Research Institute Ltd.

P - 4 & 5, C.I.T. Scheme - LXXII, Block - A, Gariahat Road, Calcutta 700 029
Phone : 440 4102/9753/9754/9847 Fax : 440 4007

35910

Dr. M. Datta

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GEMINI'S IMAGING CLINIC

131A, RASHBEHARI AVENUE

SUPARNO

CALCUTTA-700 028

PHONE : 464-1737 □ 464-3216

Miss [REDACTED] Mukherjee.

AGE 18yrs.

REFD BY Dr. A.K. Chatterjee.

REF. NO. U-1172

DATE 3.8.98

EXAMINED Whole Abdomen

ULTRA SOUND REPORT

1. LIVER: Shows smooth outline and homogeneous echo pattern. Intrahepatic ducts are not dilated. No solid or cystic mass to suggest presence of cyst, abscess, primary or secondary neoplasm. Specially no secondary deposits identified.

2. GALLBLADDER: Dilated. Shows normal smooth thin outline and cystic nature. There is no evidence of stones or mass.

3. PANCREAS: No stone identified. Calibre measures 6mm in diameter.

4. SPLEEN: Shows normal well defined outline and homogeneous echo pattern. No dilated ducts demonstrated to suggest presence of cyst, abscess or neoplasm.

5. KIDNEYS: Shows normal echo pattern.

6. ADRENALS: Not seen.

7. BLADDER: Shows normal shape, size smooth outline and cortical thickness. No evidence of calculi, nephrocalcinosis, chronic pyelonephritis, calculus, hydronephrosis, cystic neoplasm demonstrated. Cortical and medullary echo pattern is well differentiated.

8. UTERUS: Dilated. Not traceable.

9. OVARY: Not seen.

10. INTESTINES: No mass seen in bowel.

11. PROSTATE: Not seen.

12. TESTES: No residual urine remains after voiding.

13. OVARY: Not seen.

14. VAGIN: Shows smooth thin margin. There is no local recurrence of neoplasm.

(DR. M. DATTA.)




Case - 9

Anima Ganguly

I have already mentioned earlier that we have seen drastic improvements in the patients suffering from GB cancer under our non-conventional treatment. There are more than a hundred of such tissueproved patients who lived for more than five years. One of them is Mrs. Anima Ganguly, an 80-yearold lady. Her histopathology reports indicated moderately differentiated, Infiltrating Adenocarcinoma. The carcinoma had invaded the muscle coat of the gall bladder. The patient was very critical when she was first brought to us on 16/9/2008. We immediately administered Psorinum therapy along with supportive treatment. Gradually her condition improved. It has been eight years now that the patient is alive and is fit enough to do all her daily activities.

Reports and supporting documents of Anima Ganguly

DRS. TRIBEDI & ROY DIAGNOSTIC LABORATORY NABL ACCREDITED (ISO 15189 : 2003)		 Certificate No. : M0035	93, Park Street, Calcutta-700 016 Phones : 2226-6643 2226-8789 2226-8961 BRANCH : 48A, Diamond Harbour Road, Calcutta-700 027, (9 A.M. - 3 P.M.)
DR. SUBHENDU ROY MBBS. (CAL) M.D. (P.G.I.)		DR. ANITA BORGES MD. FRC. PATH (U.K.) (ASSOCIATE CONSULTANT)	
DR. A. K. BANERJEE MD. FRC. PATH (LONDON)		DR. ARJUNA RAI QUADER MBBS. (CAL) M.D. (MANIPAL)	

HISTOPATHOLOGY / CYTOPATHOLOGY REPORT			
NAME	Anima Ganguly		AGE 72 yrs. SEX F
ADDRESS			
REFERRED BY	Dr. A. N. Sen	DATE OF RECEIPT	14.05.2008
HISTOPATH. NO.	6414/2008	DATE OF REPORT	14.05.2008
MATERIAL	Histopathology slides (No. G-987) for opinion - Gall bladder		
Lab No. CKL 689			

Sections show Moderately differentiated, Infiltrating Adenocarcinoma. The carcinoma has invaded the muscle coat of the gall bladder.
No lymphovascular or perineural invasion found.

IMPRESSION :- Moderately differentiated, Infiltrating Adenocarcinoma of gall bladder.

JL DR. SUBHENDU ROY MBBS. (CAL) M.D. (P.G.I.)	L3 DR. A. K. BANERJEE MD. FRC. PATH (LONDON)	h DR. ARJUNA RAI QUADER MBBS. (CAL) M.D. (MANIPAL)
---	--	--



JANAKALYAN Nursing Home

YOUR TRUST, OUR VISION

Working Hours : Weekdays : 7A.M. to 8 P.M., Sunday : 7A.M. to 12 Noon



ULTRASONOGRAM REPORT.

Mrs. Anima Ganguly.
Dr. S. Ghosh. MBBS. (Cal)
Whole Abdomen.

70 yrs
F.
02.08.2012

Liver : It is normal in size and outlines with homogeneous echotexture and hypoechoic parenchyma. No focal cystic or solid parenchymal SOL detected. IHVS appear normal Portal vein appears normal and measures 0.71 at porta.

G. Bladder : It is not seen due to previous surgery.

Biliary T : C.B.D. appears normal and measures 0.45 cm. at porta. hepatis. Its lumen, so far visualized, appear anechoic.

Pancreas : It is normal in shape, size and outlines with homogeneous and hypoechoic Parenchyma. MPD is not dilated with no obvious detectable pancreatic pathology.

Spleen : It is normal in shape, size and outlines with homogeneous parenchyma. Its span about 5.88 cms. The splenoportal axis is patent with no detectable collaterals seen at the splenic hilum.

Kidney : Both kidney show normal in position with regular echotexture, maintaining corticomedullary differentiation. No obvious mass or calculus is seen. Right kidney measures - 7.45 cm. length. Left kidney measures - 7.61 cms. in length.

U. Bladder : It is optimally distended. Its wall thickness appears normal. No obvious intravesical mass or calculus seen. Post void residual urine volume is insignificant.

Uterus : It is anteverted and atrophied - Consistent with age.

Ad. Region : Ovaries are not delineated.

P.O.D. : There is no free fluid in POD. **Peritone :** There is no ascites.

Ret. Peri : There is no detectable retroperitoneal lymphadenopathy. Aorta and IVC appear normal.

Impression : U S G reveals - Normal study.
Please correlate clinically.

Dr. Kunal Sen,
MBBS, DMRD.

This laboratory findings, only for referral guidance, are dependent on assay procedure, equipment used & quality of sample as applicable. The final diagnosis should be made with the co-relation of other clinical findings. For any kind of technical queries relevant to this report, please contact us immediately.

27, R. N. Neogi Lane, Bhadreswar - 712 124, Hooghly, Pb : 2633 5914, M : 98301 13611



Anima Ganguly

Lung Cancer :

A would also like to discuss few specific cases of lung cancer here and to explain what I learnt from it:

After so many years of untiring hard work, now we can claim that we have been able to give some unbelievably good results. I have treated some advanced stage cancer patients who had been previously turned down by established and renowned institutions. I have tried to provide satisfactorily good service to all my patients with whatever minimum resources that I had at my disposal, at a minimum cost. On top of that I have observed considerable level of improvement in those patients under my treatment. At times it is highly disheartening to see somebody die even after pouring in my heart and soul to treat him. But this is a part of life. A few patients come to me at a certain stage when nothing further could be done. Again a few try conventional as well as every alternative treatment available to get relief from the pain that they are going through. They become very impatient and this is natural. This is the time when we should stand by the patient and his/her family.

We here basically treat patients suffering from GBM (brain tumour), lung, liver, gall bladder, stomach and pancreas, certain sarcoma and renal cell carcinoma. I have earlier answered the question that why I choose to treat these specific types of cancer. Currently at our facility-centre there are 10 beds and what all are the other infrastructure that we have, I have already discussed. We primarily treat those patients who are financially not sound. We try to provide them subsidized treatment as much as possible. On the other hand, I have treated many well-to-do patients as well. In this volume I would like to emphasize more on lung cancer and discuss some specific case studies in this context. I will always remain grateful to Prof. R. S. Bhakta and Prof. R. N. Brahmachari for guiding me through this avenue.

Among so many cases that I have treated, most of them was suffering from lung cancer. One of the reason behind this being, I had started my primary study under the guidance of Prof. Bhakta who was the professor of Chest Medicine. I had completed the total MBBS course study under his supervision, though unofficially. Along with that I had allotted some time to study lung cancer. The first 6 months I had to work very hard to understand the anatomy and physiology of chest. Prof. Bhakta then advised me to study all the diseases and infections related to lungs in the next 6 months. He also taught me the management of certain critical cases. At times he used to discuss his personal experiences in this front and a few complicated cases he had come across. But he told me that he never treated someone suffering from lung cancer. He used to tell me the various differences between lung cancer and all other diseases and infections related to lungs. He also helped me to distinguish between the x-ray plates of a lung cancer patient and a person suffering from tuberculosis. In due course I had acquired a fair idea of diseases related to lungs. There are many types to lung cancer and so much of diversity and complexity associated with it that it becomes very difficult at times to distinguish one from the other. For a more detailed knowledge on lungs cancer he asked me to remain in constant touch with Prof. Brahmachari as he was an oncologist and on certain

occasions we referred to him. However, within 2.5 years I was able to go to the depth of the subject and understood the intricacies related to it. Gradually things became quite easy for me.

One day I went to Prof. Bhakta to express my thankfulness that because of him and Prof. Brahmachari I learnt so much on the subject of lung cancer. Prof. Bhakta in return said that in order to answer my questions he had to study a lot and in due course came across certain realisations and facts that were also unknown to him. He appreciated me stating that in a short span of time I was absorbed so much in learning under Prof. Brahmachari's and his supervision. Prof. Bhakta said: ***Sometimes I used think that you are learning too much. This might be unhealthy. But soon I realised that everything was going as it was supposed to. I accept that at times I become short-tempered and I have also scolded you so many times. But you always took it on a positive note.*** He further added that: ***In my later life if someone asks me who my best student is. Without any hesitation my answer will be Asim.***

Today's generation who are treating lung diseases, cannot imagine that up to early 1990s how difficult it was to detect diseases like pneumonia, tuberculosis and lung cancer. In order to detect lung cancer a hard tube had to be inserted and passed through the lungs. It was so difficult even to perform Bronchoscopy. It was not easy to identify tuberculoma, fungal infection and hydatidosis by looking at the x-ray plates. During these days the two renowned and experienced medical practitioners, Prof. R. S. Bhakta and Prof. R. N. Brahmachari, guided me throughout and made me practise in these tough conditions to show me the real picture.

One day Prof. Bhakta was teaching me on the topic viz. pancoast carcinoma. I was having difficulty in understanding it. During that time a patient came up to Prof. Bhakta with the complain of severe chest pain. The pain had spread to one of his hands as well. One night suddenly Prof. Bhakta called me at around 10:30 telling me to get ready, as we were going to visit that patient at his residence in Belghoria. That day Prof. Bhakta himself was suffering from high viral fever, still he went to visit that patient. He picked me up from my residence within 15 minutes. When he examined the patient, he immediately realised that the patient was suffering from pancoast carcinoma. He then attended that patient and we returned back home by 2 a.m. that day. The next seven days he taught me about pancoast carcinoma and how it is different from the other types of carcinomas:

A Pancoast tumor, also called a pulmonary sulcus tumor or superior sulcus tumor, is a tumor of the pulmonary apex. It is a type of lung cancer defined primarily by its location situated at the extreme apex of either the right or left lung. It typically spreads to nearby tissues such as the ribs and vertebrae.

Pancoast tumours are named for Henry Pancoast, a US radiologist, who described them in 1924 and 1932.

This syndrome results from the invasion of a number of structures and tissues around the thoracic inlet and aside from cancer general symptoms such as malaise, fever, weight loss and fatigue, Pancoast tumour can include a complete Horner's syndrome in severe cases: miosis

(constriction of the pupils), anhidrosis (lack of sweating), ptosis (drooping of the eyelid) and enophthalmos (sunken eyeball). Although a Pancoast tumour is a lung tumour, it rarely causes symptoms that are typically related to the lungs (e.g. cough, chest pain). The initial symptom is pain in the shoulder, inner part of the scapula (large, triangular, flattened bone that lies over the ribs on the back), or both. In progressive cases, the brachial plexus is also affected, causing pain and weakness in the muscles of the arm and hand (brachial plexus invasion C8-T2) leading to wasting of the intrinsic hand muscles and paraesthesiae (a sensation of pricking, tingling or creeping on the skin) in the medial side of the arm. The tumour can also compress the recurrent laryngeal nerve producing unilateral vocal cord paralysis and from this a hoarse voice and bovine cough may occur, and/or phrenic nerve involvement. In superior vena cava syndrome, obstruction of the superior vena cava by a tumour (mass effect) causes facial swelling cyanosis and dilatation of the veins of the head and neck.

In as many as 10%-25% of people with Pancoast tumour, compression of the spinal cord and paraplegia (paralysis of the lower half of the body with involvement of both legs) develop when the tumour extends into the intervertebral foramina (opening between two vertebrae).

Other tumours can also result in Pancoast's syndrome eg, breast cancer, mesothelioma, plasmacytoma or lymphoma; or metastatic carcinoma - eg, from the larynx, cervix, bladder, thyroid or colon).

Non-neoplastic causes of Pancoast's syndrome are very rare - but there have been reported cases due to bacterial pneumonia (staphylococcal or pseudomonas), tuberculosis, hydatid disease, mycotic aneurysm, disseminated nocardiosis and plasma-cell granulomas. It has also been reported as due to non-Hodgkin's lymphoma, cervical rib and pulmonary amyloidosis.

The risk factors for almost all lung cancers are similar. These include:

- Smoking
- Secondary smoke exposure
- Prolonged asbestos exposure
- Exposure to industrial elements (e.g. gold, nickel).

Pancoast tumours may affect other parts of the body:

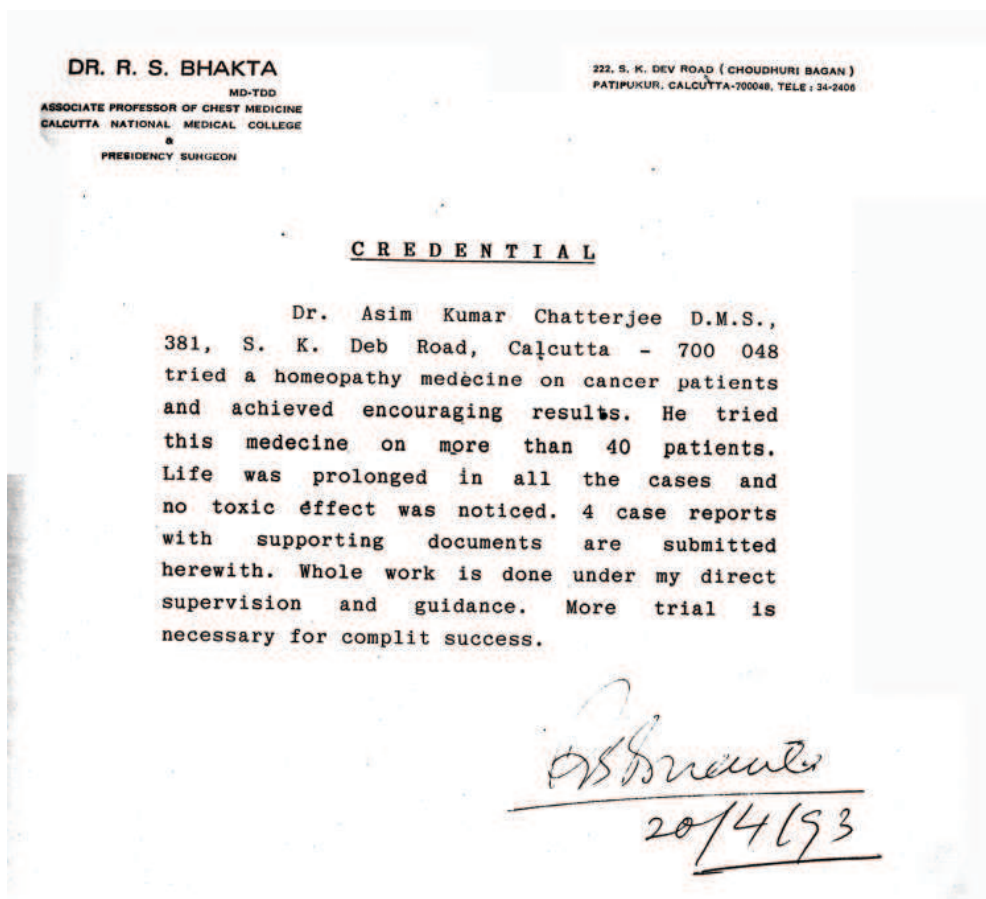
- Lymphatics (small, thin vessels that carry lymph fluid through the body)
- Lower roots of the brachial plexus (a complex network of nerves that is formed chiefly by the lower four cervical [neck] nerves and the first thoracic [chest] nerve)
- Intercostal nerves (nerves that lie between a pair of adjacent ribs)
- Stellate ganglion (a mass of nerve tissue containing nerve cells that form an enlargement on a nerve or on two or more nerves at their point of junction or separation)
- Sympathetic chain (either of the pair of ganglionated lengthwise cords of the sympathetic nervous system that are situated on each side of the spinal column)
- Adjacent ribs

- *Vertebrae.*

The treatment of a Pancoast lung cancer may differ from that of other types of non-small cell lung cancer. Its position and close proximity to vital structures (such as nerves and spine) may make surgery difficult. As a result, and depending on the stage of the cancer, treatment may involve radiation and chemotherapy given prior to surgery (neoadjuvant treatment). Surgery may consist of the removal of the upper lobe of a lung together with its associated structures (subclavian artery, vein, branches of the brachial plexus, ribs and vertebral bodies), as well as mediastinal lymphadenectomy. Surgical access may be via thoracotomy from the back[2] or the front of the chest and modifications.

As long as the cancer has not metastasised (spread) and involved the regional lymph nodes (small, bean-shaped structures found throughout the body), these tumours can be successfully treated.

Prof. Bhakta then asked me whether I would be able to give some relief to the unbelievable pain the patient was going through. I said that I would try my level best. Fortunately I was able to give some relief to the patient. The patient survived for around two years. Unfortunately the patient's documents are not available with me but owing to this case, a good bond developed between me and Prof. Bhakta and I will cherish it my entire life.



The case studies I discuss here relate to the patients who come from various socio-economic classes and from varied age groups. I have intent to give a success-table of the cases I have treated in the next volume of this book receiving joint co-operation from Prof. Bhakta, Prof. Brahmachari, Prof. Jaydeep Biswas and Prof. Anup Majumdar. 6 cases are worth-mentioning here:

Case No. 10

Shanti Ranjan Pal (Redg. No.: CCMRCC/0153/2009-10) is a 65 year old patient coming from an economically backward family. On 15.10.2009 he visited a local doctor following a severe chest pain and haemoptysis. The local doctor advised them to undergo CT Scan. On 24.10.2009 the CT Scan was conducted which showed left lung SOL – neoplastic, focal pleural thickening on the right and emphysematous changes in both lung fields. FNAC was conducted (from the DRS. Tribedi and Roy) and the report on his lung SOL suggested moderately differentiated Adenocarcinoma in the left lung. The patient got admitted in the Radiotherapy department of the Medical College. The doctors there suggested for Radiation and Chemotherapy (RT & CT). However, the patient could not afford it. The patient got admitted at our Critical Cancer Management Research Centre & Clinic (CCMRCC) on 5.11.2009. We started administering Psorinum Therapy from the very next day. Sometimes without conducting RT & CT, while symptomatic anti-biotic medicines were also given to him. Along with Psorinum Therapy supportive treatment by Prof. Anup Majumdar, Prof. Amitabha Chakraborty and Dr. Hiranmoy Mukherjee was also provided. On 16.5.2015 the patient was admitted at the CCMRCC for the second time. Due to the patient's critical condition x-ray or CT Scan could not be conducted. The patient died on 21.5.2015 in Critical Cancer Management Research Centre. Finally we were unable to detect that whether cancer had reoccurred or the patient died for some other reason.

Reports and supporting documents of Shanti Ranjan Pal

Dr. Debasis Banerjee MD

Ref. No : 6373 DATE : 24/10/09

NAME : SANTI RANJAN PAUL

AGE : 65 Yrs. SEX : MALE

Referred By : Dr. of M.C.H.

M.R.No. : 10554

REPORT ON FINE NEEDLE ASPIRATION CYTOLOGY

SPECIMEN : C.T. guided FNA from S.O.L. In apex of left lung.
(Unstained smears are received).

MICROSCOPIC EXAMINATION :

Smears show a few clusters of epithelial cells with nucleomegaly, nuclear pleomorphism, hyperchromasia, high nuclear-cytoplasmic ratio and pale cytoplasm. Nucleoli are visible in some of the cells.

REMARKS : Non small cell carcinoma.

Enclosed : Slides


Dr. Debasis Banerjee MD

Clinic and Laboratory

Clinical Haematology Service
4, Gorky terrace, Kolkata-700 017
Ph.: 22837471, 22836629, Fax : (033) 280-1807
Email : clinical.haematology@vsnl.net

NIRIKSHAN
Pathological laboratory Pvt. Ltd.
40, Dobson Road, Howrah-711 101
Phone : 2676 - 9816

SANTI RANJAN PAUL**65 YRS****24.10.2009**

005

DR. OF MCH**C T SCAN OF THE CHEST****TECHNIQUE**

Plain & I.V. (non -ionic) contrast enhanced C T scan of the chest done with 5 mm. and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the chest in supine position and in frontal projection reveals nodular opacity in the left upper zone.

Bones under review shows no detectable abnormality. Parietal muscles appear normal. No sizeable mass is detected in the axillae.

Great vessels of the mediastinum including ascending aorta, arch of aorta and its branches, descending aorta, M.P.A. and its branches, S.V.C. and its tributaries appear normal. Trachea and its bifurcation are normal. No sizeable mass is detected in the mediastinum.

Focal pleural thickening seen on the right. Left pleural space is normal.

An irregular heterogeneously enhancing non-calcified S.O.L. (33 x 26 x 30mm) is seen at the left upper zone, in close contact with the pleura. Emphysematous blebs are seen in both lung fields.

Contd2..

CT & MRI SCAN CENTRE



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Dept. of Health & Family Welfare, Govt. of West Bengal)

-2-

SANTI RANJAN PAUL

65 YRS

24.10.2009

IMPRESSION

- Left lung S.O.L. – neoplastic.
- Focal pleural thickening on the right.
- Emphysematous changes in both lung fields.

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD


DR.SOUMITRA SEAL
MD

DR.D.SHARMA
MD

DR.ANUP SADHU
DMRD, MD.

DRS. TRIBEDI & ROY
DIAGNOSTIC LABORATORY
NABL ACCREDITED (ISO 15189 : 2007)



Certificate No. : M0035

93, Park Street, Kolkata-700 016
Phones : 2226-6643
2226-8789
2226-5961

BRANCH : 48A, Diamond Harbour Road,
Kolkata-700 027, (9 A.M. - 3 P.M.)

NAME Santi Ranjan Paul 65 yrs. (Lab No. DBS 1036)
ADDRESS _____ DATE OF RECEIPT 27.10.2009
PHYSICIAN Dr. Of M. C. H. DATE OF REPORT 29.10.2009
MATERIAL FNAC slides for review

FNAC SLIDES FOR REVIEW

Received three stained slides No. 6373 representing CT guided FNAC from
(Left) Lung SOL.

Stained smears show clusters and discretely present pleomorphic malignant cells
having large hyperchromatic nuclei and variable amount of cytoplasm.

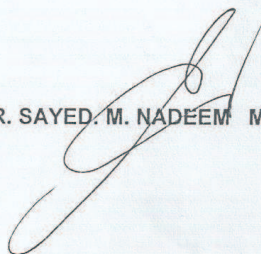
DIAGNOSIS :-

(Left) Lung ---- Moderately Differentiated Adenocarcinoma.

F/3011/09

SMEAR : 03

DR. SAYED. M. NADEEM M.D

A handwritten signature in black ink, appearing to be 'Sayed M. Nadeem', written over the printed name.

DEPARTMENT OF HEALTH AND FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
DISCHARGE
Medical College & Hospital, Kolkata
88, College Street, P.S. : ,

Friday MORND
UTC: Unit 2 AM.

Discharge Certificate/Left Against Medical Advice

Page No. : 1

Discharge No. : Date of Discharge : 28/10/09 Time : 4:00 pm. Patient Category : Free / Paying / Cabin
Patient Name : SANTI RANJAN PAL Sex : Male Age : 66 Yrs. 6 Months 6 Days 6 Hrs
Patient Srl. No. : Patient Registration No. : Admission Date : 16-Oct-2009 Time: 19:36
Address : PA09102007 RG#9399269
Municipality / Village : 65A, SOVA BAZAR ST Post Office : HATKHOLA
Police Station : Shyampur P.S. District : Kolkata
State : West Bengal Nationality : Indian Religion : Hindu Other :
Father's Name : LT/H.K. PAL Free Husband's Name :
Doctor/Unit : 229 DOC2401 PROF. DR. BISWAS/ DR. S. PAI/DR. B. GHOSH Phone/Mobile No. :
Admission No. : 229 DOC2401 Ward Name :
Final Diagnosis : Non small cell lung carcinoma & BHP M.C.H. 2ND Floor (Male) (IPW11)

Referred To : Medical Oncology Dept, MCH. Time : Reason :
② CNCR.

In case of Confinement
Delivery Date & Time : Mode Of Delivery : ND/ECL/LUCS/With Forceps/Without Forceps
Delivery Status : No. Of Child : Antenatal Care Taken : Yes / No

In case of Surgery
Surgery Date & Time : Type of Surgery : Details of Baby
Surgery Status : Birth Date : Birth Time :
Disc No. : Sex :
Birth Wt. :
Advice for Baby

Tests done:
S/UTC - 125/38/10.8 Anesthesia - 104/3.22
CH: 46-15.2 TC-9800 (N92 L6 B2) MEV - 29.9 MCH-28. MCHC-35.2
Platelet - 1.36 lakh/cmm
Urine R/B : WNL

Investigation Done
Test No. 22/10 LFT: Bilu-0.7 SGOT-54 SGPT-46
ALP-221
Sr Cat+ - 8%
Na+ - 117, K+ - 2.89
29/10 Na+ 126.2/5.17.


Medicine Name No. of Days Details Comments
23/10 Na+ - 120.6
3% NaCl T Urinax
T Stenilo Syr Potklor
w/ Ceftriaxone
w/ Rantel

ADVICE
① Normal diet & add salt 1 tbf BD x lmt
② T Urinax (0.4 mg) 1 tab at bed time: x lmt
③ T Stenilo (5) - 1 tab BD x lmt
④ Prostate Biopsy
⑤ Review of PNAE slide
⑥ To attend Medical Oncology

Counter Signature of the Visiting Staff
OPD & above mentioned reports for further follow up or attend ER MS.
Signature of the Medical Officer: 28/10/09

Case discussion

60 y known smoker and hypertensive male presented with drowsy disoriented state following repeated vomiting. He also had high grade fever & chill + rigor. He had above U/O frequency & urgency of urination. He had clubbing, hypertension, generalized rigidity. Falciticks kit (-ve). He was immediately catheterised for urinary retention. Severe symptomatic hyponatraemia was documented and 3% NaCl was given & proper monitoring. CXR showed well defined shadow in left apical lung field, CT thorax confirmed neoplastic lesion and PNAAC documented non-small cell lung carcinoma. USG documented BHP, PSA was markedly raised. Patient advised for rectal biopsy and to attend medical oncology OPD for further follow up. सत्यमेव जयते


28/10/09

Department of Health & Family Welfare
Government of West Bengal

Case no.: 11

Kashinath Saha, a 51 years old poor worker of a locked-out company, in January 2001 complained of severe chest pain and haemoptysis. The x-ray suggested a homogenous opacity seen in the right lung. A CT Scan of the thorax and FNAC were conducted on 12.3.2001. The FNAC report showed Adenocarcinoma of the lung. From the radiotherapy department of the Medical College under Prof. Subir Ganguly a medical card was made No. 51/H/M and 2001/713 on 17.4.2001. He was advised to undergo Chemotherapy. The patient expressed his inability to afford Chemotherapy sessions. 25.4.2001 onwards Psorinum Therapy was administered to the patient. Gradually the patient's condition improved and within one year his total right lung mass regressed. For the next five years the follow-up of the patient was done at the radiotherapy department of the Medical College. The patient till date is fine and is leading a healthy life and now running a small tea and beetle shop. When some of our cases were reviewed by the famous National Institute of Cancer, USA, Kashinath Saha's case was one of them. During his treatment the patient's neighbours and friends for around one and a half years helped him and his family in several ways.

Reports and supporting documents of Kashinath Saha



NORTH CITY

DEPARTMENT OF PATHOLOGY

Regd. No. CYTO/13849

19.03.2001

=====

Patient's Name : MR. KASHINATH SAHA Age : 51 Y Sex: M |

| Refd. By : DR. S. K. SAHA Date : 17.03.2001 |

=====

REPORT ON FINE NEEDLE ASPIRATION CYTOLOGY

SITE OF ASPIRATION : FNAC of Right upper lobe of lung.

MICROSCOPIC DESCRIPTION:-

The smears are composed of malignant epithelial cells. These cells are pleomorphic with anisonucleosis and fair amount of cytoplasm. They are arranged in sheet and acinar pattern.

DIAGNOSIS:- ADENOCARCINOMA.

ENCLOSED :- One stained slide. (FNAC NO - F/131/01)

DR. (PROF) M. CHAKRABORTY
MD (PATH & BACT)
CONSULTANT PATHOLOGIST

DR (Col) A. PANDIT
MD (PATH & BACT), D C P
CONSULTANT PATHOLOGIST

North City Diagnostic & Research Centre 35A, Canal West Road, Calcutta - 700 004,
(Near Gouri Bari Bus Stop) Tel : 554 6221/6225/543 2374
From 7.00 a.m. To 8.00 p.m. (Mon - Sat) From 7.00 a.m. To 2.00 p.m. (Sunday)

(৪)

RADIO THERAPY DEPT.

2004/713

এক টাকা

তারিখ

চিকিৎসার বিবরণ

DR. SUBIR GANGADHYA

Radiotherapy Department
Medical College Hospital, Calcutta.RADIO THERAPY DEPT.
UNIT - I
DR. SUBIR GANGADHYA
মহাশয়, প্রত্যাশিত.

হাসপাতালের বহির্বিভাগে রোগীদের চিকিৎসার জন্য টিকিট

নং 511557

তারিখ: 17-4-2004

M. C.

হাসপাতাল

নাম:

Kashi Nath Saha

বয়স:

51

পুরুষ/মহিলা:

H

রোগের নাম:

CA Lung (R)

তারিখ

চিকিৎসার বিবরণ

17/4/04

1st - 2nd 45Gy.
 Radiotherapy
 CPT
 1st & 2nd 45Gy.

To come to the report
 for consideration
 of chemotherapy.
 Signature

B.C.L. / Ticket / M-S / 30,00,000 / 2k

	(২)	(৩)
তারিখ	চিকিৎসার বিবরণ	তারিখ
17/6/03	<p> 17/6/03 Patient Pati 2 years all on homeopathy tree hut L. Scan 2 Chir in a Rump Can of Ca lip Rept 12/3 </p>	

RADIOTHERAPY DEPT.

UNIT - IB

মঙ্গল-বৃহস্পতি

Form No. 42 Medical College Hospitals, Cal

Medical College Hospitals

Department of Radiology (Therapeutic)

CALCUTTA

Name, Kashi Nath Saha - 51/H/M
Number, 2001/713
Date of 1st Attendance, 17.4.2001

Please bring this Card with you when attending Hospital. If under to attend on the date stated another appointment should be made.

হাসপাতালে আসিবার সময় এই কার্ড আনতে
ভুলবেন না। নির্ধারিত দিনে না আসিতে পারিলে অন্য
দিন ঠিক করিয়া লইবেন।

असपाताल मे जाने के समय यह कार्ड लाने को न भुलिये।
नर्धारित समय पर अगर न आने सके तो दूसरी तारीख के लिये
लिख भेजिये।

Ca Lung (Rt)

EKO CT SCAN CENTRE

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Dept. of Health & Family Welfare, Govt. of West Bengal)



KASHI NATH SAHA

51 YRS

21.03.2003

DR. SUBIR GANGOPADHYAY

CT SCAN OF THE CHEST

HISTORY

Follow up patient of adenocarcinoma right lung - FNAC on 17.03.2001 - at present doing well.

TECHNIQUE

Plain & I.V. (non-ionic) contrast enhanced C T scan of the chest done with 5 mm. and 10 mm. sections in the axial plane.

FINDINGS

Digital radiograph of the chest in supine position and in frontal projection shows collapse at right upper lobe.

Bones under review shows no detectable abnormality. Parietal muscles appear normal. No sizeable mass is seen in the axillae in this scan study.

Mild pleural thickening seen at upper part of right lung.

Great vessels of the mediastinum including ascending aorta, arch of aorta and its branches, descending aorta, M.P.A. and its branches, S.V.C. and its tributaries appear normal. Cardiac outline is normal. Trachea is pulled to right. No sizeable mass is detected in the mediastinum.

Lung parenchyma - Collapse with bronchiectatic changes & fibrosis involving right upper zone. Rest of the lung fields are clear.

Contd ...2..

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-2-

K.N.SAHA

(CT SCAN OF CHEST)

21.03.2003


IMPRESSION

Present C.T. features are suggestive of collapse fibrosis with bronchiectatic changes involving right upper lobe.

Suggested clinical correlation and further investigations if clinically indicated.

DR.S.K.SHARMA
MD

DR.SOUMITRA SEAL
MD


DR.ANUP SADHU
MD, DMRD

AT Medical College & Hospitals Campus
88, College Street, Kolkata - 700 073, Phone : 2219 - 7511/2219 - 7512




Kashinath Saha with Dr. Jeffery White

Case no.: 12

Dilip Kumar Pal, 61 years old, a worker of a lock-out jute mill. He was suffering from severe chest pain and also haemoptysis. His first CT Scan was conducted on 19.4.2014. The reports suggested a left upper lobe mass with mediastinal invasion and mediastinal lymphadenopathy. FNAC report suggested right-sided mediastinal mass. Metastatic non-small cell Adenocarcinoma was diagnosed. Without any more delay they came to us and Psorinum Therapy was administered. Till date the patient is in quite a good health.


Reports and supporting documents of Dilip Kumar Pal

EKO CT & MRI SCAN CENTRE		
A UNIT OF EKO DIAGNOSTIC PVT. LTD. (A Joint Venture with Eko Diagnostic Pvt. Ltd. & Dept. of Health & Family Welfare, Govt. of West Bengal)		
DILIP KUMAR PAUL	62 YRS	17.07.2014
DR. OF MCH		005
<u>CT SCAN OF THE CHEST</u>		
<u>HISTORY</u>		
Follow up patient of Ca.lung (right) – last CT 19/4/14.		
<u>TECHNIQUE</u>		
Plain & I.V. (non –ionic) contrast enhanced C T scan of the chest done with 5 mm. and 10 mm. sections in the axial plane.		
<u>FINDINGS</u>		
Digital radiograph shows right hilar opacity.		
Bones under review shows no detectable abnormality. Parietal muscles appear normal. No sizeable mass is detected in the axillae.		
Great vessels of the mediastinum including ascending aorta, arch of aorta and its branches, descending aorta, M.P.A. and its branches, S.V.C. and its tributaries appear normal. Trachea and its bifurcation are normal. Right hilar lymphadenopathy shown.		
Irregular mass (59 x 27 mm) seen in right parahilar area.		
No evidence of any pleural thickening or pleural effusion is seen.		
Contd2..		
AT Medical College & Hospital Campus 88, College Street, Kolkata - 700 073, Phone : 2212-3778/3779		

Case no.: 13

Chandi Charan Rhuidas, a 58 year old cobbler, was complaining of chest pain and haemoptysis. He was admitted at SSKM Hospital under the treatment of Dr. S. Basu (Registration No.: RT/M/763/14). He was advised to undergo chemotherapy. Due to his financial inability he refused to administer chemotherapy and came to us at the end of April. We started his treatment with Psorinum Therapy the very next day. Till date he is alive and working.

Reports and supporting documents of Chandi Charan Rhuidas

WINMARK Ph: (033) 22865737		WINMARK DIAGNOSTIC SERVICES (A joint Venture with Govt. of W.B.) Campus: Calcutta National Medical College and Hospital, Kolkata 700 014
Patient ID : 250314/08		Date: 25.03.2014
Name: Chandi Charan Ruhi Das		Age: 57 Years Sex: M
Ref. By : Dr. D.P. Dolui		

C.E.C.T. SCAN OF THORAX

PROCEDURE :
Multidetector CECT Scan of chest was done in axial plane (with coronal reconstruction) from lung apex through domes of the diaphragm by taking 5 mm thin contiguous parallel slices.

DIGITAL RADIOGRAPH :
On the thorax frontal projection show a rounded soft tissue opacity in right upper mid zone.

BONES :
Under review show no obvious lesion. Parietal and Paravertebral muscles are seen normal. No sizeable mass is seen in either axillae.

PLEURA :
No significant lesion is seen on either side.

MEDIASTINUM :
Vessels in the mediastinum show no obvious lesion. Trachea and oesophagus are unremarkable. Cardiac silhouette is normal. Tiny discrete lymphodal enlargement is seen in pretracheal and aortopulmonary window location.

LUNG PARENCHYMA :
No significant focal parenchymal lesion is evident in left lung. A well defined, large, heterogeneously enhancing. Soft tissue attenuating mass lesion (10.9 x 10.5 x 9.1 cm) is noted in right upper lobe, abutting the posterolateral chest wall and stretching and partially occluding the right upper lobar bronchus. No underlying rib erosion is noted. Rest of the lung parenchyma appears normal. No evidence of bronchiectasis is seen.

Contd.....

WINMARK

Ph: (033) 22865737



WINMARK DIAGNOSTIC SERVICES

(A joint Venture with Govt. of W.B.)

Campus: Calcutta National Medical

College and Hospital, Kolkata 700 014

Patient ID : 250314/08

Date: 25.03.2014

Name: Chandi Charan Ruhi Das

Age: 57 Years Sex:M

Ref. By : Dr. D.P.Dolui

RESSION:

1). A well defined, large, heterogenously enhancing. Soft tissue attenuating mass lesion is noted in right upper lobe, abutting the posterolateral chest wall and stretching and partially occluding the right upper lobar bronchus. No underlying rib erosion is noted. CT guided FNAC is suggested from the mass.

2). Tiny discrete lymphnodal enlargement is seen in pretracheal and aortopulmonary window location.

Sanjuka

DR. SANJUKTA SARKAR

MF. RADIOLOGIST, FRCR(U K)

Signature



DEPARTMENT OF HEALTH & FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
OPD Patient Card

S.S.K.M. HOSPITAL & IPGME & R
A.J.C. BOSE ROAD, KOLKATA-20

Dr. S. BASU
TUESDAY / FRIDAY
মঙ্গলবার / শুক্রবার
RT / M / 763 /

Name :	CHANDI CHARAN RUIDAS	[ORI14169978/1]	Day :	Tuesday	Paid Rupees 2.00
Sex :	Male	Age : 58 Yrs.	Months	Days	Reg. No. : RG14205746
Ref. From :					Reg. Date : 01-Apr-2014
					Card No. : ORI14169978/1
Visit No. :	1	Department RadioTherapy OPD	Visit Date :	01-Apr-2014	Time : 12:51
Doctor/Unit Name (DOW)	Dr. SIDDHARTHA BASU (Tuesday/Friday)	[Friday/Tuesday]			
Room No.	17		Entry No. :		

Visit Date :	Visit No. : 2
Department :	Tm.
Doctor/Unit :	
Entry No. :	01 APR 2014

Visit Date :	Visit No. : 3
Department :	Tm.
Doctor/Unit :	
Entry No. :	

Visit Date :	Visit No. : 4
Department :	Tm.
Doctor/Unit :	
Entry No. :	

Clinical Notes	ADVICE
<p>- C/o persistent cough since last 7-8 mths.</p> <p>- 2 episodes of haemoptysis since last month.</p> <p>- Stigab chestpain since last month.</p> <p>↓</p> <p>No palpable axillary / Supraclav. L. Nodes B/L.</p> <p>↓</p> <p>Ex Thorax: 11.0 x 9.1 x 9.1 cm</p> <p>mass in R upper lobe</p> <p>Any discrete L. Nodal enlargement in pretracheal and aortopulm. window</p>	<p><u>Aclis</u></p> <p>- Blood for Ur/Cr/ Su(A)</p> <p>Total Bil / SuOT / SuPT</p> <p>Alkaline Phosph</p> <p>HIV I & II, HBSAg</p> <p>Serum Uric Acid</p> <p>- USG Whole Abdomen.</p> <p>- To attend CTVS OPD for surgical opinion</p> <p>- To attend RTU OPD</p> <p>Go with defaults in Tues / Fri / ERSC</p> <p>→ Ibuprofen (400)</p> <p>Tab Durango - BDPC } 5 days</p> <p>Tab Ramlate - BDAc (150)</p>

Prof. Dr. D. Paul, Hon.
Tuesday & 1st Sat.
Chest OPD. Unit 1A

DEPARTMENT OF HEALTH & FAMILY WELFARE
GOVERNMENT OF WEST BENGAL
OPD Patient Card

01 APR 2014

S.S.K.M. HOSPITAL & IPGME & R
A.J.C. BOSE ROAD, KOLKATA-20

Name :	CHANDI CHARM RUDAS	[DR14169675/1] Day :	Tuesday	Paid Rupees 2.00
Sex :	Male	Age :	38 Yrs.	Months Days
Ref. From :		Reg. No. :	RS14205322	
		Reg. Date :	01-Apr-2014	
		Card No. :	DR14169675/1	
Visit No. :	1	Department :	Chest OPD	
Doctor/Unit Name (DOW) :	Dr. D. Pal (Unit 1-A) (Chest)/1st sat/tuesday (Tuesday)	Visit Date :	01-Apr-2014	Time : 11:56
Room No. :	15	Entry No. :		

Visit Date :	Visit No. : 2	Visit Date :	Visit No. : 3	Visit Date :	Visit No. : 4
Department :	Tm.	Department :	Tm.	Department :	Tm.
Doctor/Unit :		Doctor/Unit :		Doctor/Unit :	
Entry No. :		Entry No. :		Entry No. :	

Clinical Notes	ADVICE
<p>no cough & cold</p> <p>c' chest pain > P-10 mm</p> <p>Re-laminar (P-20) S & 2</p> <p>Hypomelic</p> <p>MC h- dea</p> <p>CT thum</p> <p>a well defined large</p> <p>heterogeneous enhancing</p> <p>soft tissue attenuating</p> <p>mass (area RUL)</p> <p>tiny discrete lymphovascular</p> <p>enlargement peripheral</p> <p>subpleural windows breaks.</p> <p>Wash & 2</p> <p>DOTS m 24/3/14</p> <p>From @ hr son</p> <p>= Schinoc</p>	<p><u>Ref</u></p> <p>Ref to RTU and</p> <p>02/04/14</p>

Many poor patients like a few I have mentioned here are undergoing Psorinum Therapy and leading a good health.

Parul Bala Dey's cases fall under the scope of lung cancer which I have already discussed and Gobindo Das Ghosh and Nanda Rani Banerjee's cases are documented in the paper titled 'Psorinum Makes a Major Break Through in the Treatment of Tobacco Related Lung Cancer' which I have presented earlier.

In this context the reader can also refer to an article documented in the Journal of Thoracic Oncology, Volume 5, Number 12, Supplement 7, December 2010, titled 'Psorinum Therapy in Treating Patients With Advanced Non-small Cell Lung Carcinoma (NSCLC): A Phase-II Single Arm Clinical Trial' by (Dr. Aradeep Chatterjee) which I have presented in the later part of the book.

We always keep an eye on, try to help and treat, socially backward people who are either unemployed poor or retrenched workers of closed mills and factories. The patients as well as their families require proper counselling before and during the treatment to infuse a new desire to live among the patients who have lost all hopes. We also try to minimise the cost of treatment as much as possible and make things more affordable to them allowing them many concessions and providing medicines at subsidised rates. Along with this when we add Psorinum Therapy we get pretty good results.

The four cases that I have mentioned here viz. Shanti Ranjan Pal, Dilip Kumar Pal, Chandi Charan Rhuidas and Kashinath Saha where the patient came from a similar background.

When I decided to embark on this journey I had to put in a lot of effort to achieve this goal. However, I never faced any severe resistance that ever halted my activities and research. Working with Prof. R. N. Brahmachari and Prof. Anup Majumdar at Cure Point Nursing Home I never had any issues with these eminent personalities because they were always my well-wishers and my guides in my journey. But they had also advised me that in order to bring about any institutional change I would to break the existing structure and recreate it. This entire process was going to be very difficult but not impossible. Fortunately I was able to break many barriers in my journey so far, and that might be because of the fact that I always had sufficient finances at my disposal. Prior to this for two years I had worked under very complicated situations and that experience helped me in the long run. I had also chosen the famous cancer institutions of India to carry forward with my work like Chittaranjan National Cancer Institute, SSKM Hospital PG, Department of Radiotherapy of Medical College, the School of Tropical Medicine, Thakurpukur Cancer Hospital, R. G. Kar Medical College and Hospital and Tata Memorial Hospital (Bombay) and a few renowned personalities in the medical community that I wanted to work with. I knew I would need to do something that the world had not witnessed ever.

In my childhood days I used to play football. I used to notice a small boy watching our match. One day he entered the field, took the ball, carried it well and scored a goal. Like that

small boy I had to learn the game in the field, while playing it and had to score the goal as well. I was quite fortunate to achieve that as well.

If you go through these case studies you would realise that I had to overcome so many odds as well as legal barriers in my way. I had done all my works in such a way that the institutional heads would give their consent and show acceptance towards my work. I have come so far not for me but for the cancer victims. My study is dedicated towards them. My intention is not to disrespect someone but to benefit the entire mankind. If you find me wrong anywhere, punish me; if not, give me hands to serve humanity and for the greater good.

WHAT OTHER DOCTORS SPEAK ABOUT DR. ASIM CHATTERJEE

– By Piyanka Mohanty

Conversation with Dr. Anup Sadhu

How and when did you come to know Dr. Asim Chatterjee?

I have heard about him since 1998-99. At that time, I was not in Kolkata. I was connected to Medical College directly since 2000-2002. From then on I came in direct contact with Dr. Asim Chatterjee. Many doctors of Radiological Department in Medical College send many cancer patients to Dr. Chatterjee. Initially, I was surprised. Later, I came to know that he treats many cancer patients through Psorinum Therapy and many people are quite well. Since then I got close to him. I observed that many doctors are sending such patients to him who even could not undergo any diagnosis. His medicine along with all required palliative treatment is improving conditions of many CT report of patients treated here show no further enlargement of Tumour. In many cases it has been no deterioration of patient's condition. I personally have studied many cases. Psorinum Therapy is quite helpful to keep patients stable.

How did you get involved with Dr. Chatterjee in cancer projects?

Actually, he is a very simple person. He has never says to anyone that nothing can be done, he always says that he will try his best. But he never provides false promises to others. He has always provided courage to the patient family and has always cooperated with them.

When Dr. Chatterjee used to go to Subodh Mitra Cancer Hospital, once he took me late minister Subhas Chakraborty over there. He was aware of my work and praised our work.

At that time, Dr. Chatterjee and others were arranging for treatment and accommodation of many patients of Subodh Mitra Cancer Hospital. Economical arrangement for accommodation of cancer patients could be provided through Gandhi Seva Sangha. Even travelling to and from Subodh Mitra was quite easy from and to there. Patients from abroad, coming here for Radiation could stay there. But he found some problem to continue working in Subodh Mitra. So he developed a Palliative Management Centre in his residence only.

What would you say about effectiveness of Psorinum Therapy?

Generally cancer treatment is quite expensive. This is because the drugs are quite costly. Demand cannot be fulfilled even with the government subsidy. I gave my opinion as I came close to Dr. Chatterjee. Tried to explain how cancer can be treated at lesser cost through Palliative management, and I also tried to show how can pain management and other complexities of cancer be dealt at even 5 times lesser cost.

Besides these, we published CT Scan, FNAC guided differrent journals. Gradually, these issues came out in even international papers. I have many patients here who regularly consume Dr. Chatterjee's medicines and are very fine. One man survived even for 4years with

Colon cancer. He used to walk from Sealdah to come here. Even patients in 4th stage of Liver, Lung, Pancreas, Gall bladder Cancer are much relieved from pain by his medicines. I have sent many of my relatives to him and they also felt benefited.

It is not possible now to say directly about the working procedure of his medicine. But we have observed that it works according to specific dose. Even, a few years ago, a project work was started at IIT Kharagpur to find out how a particular molecule of this drug is working on a cancer cell. But there were some complications regarding the clearance of the project. So it had to be rejected. There has been attempt to find out details like – how is the therapy working on Stage -1, Stage-2 and Stage- 3 patients, if there are any side effects, how is one particular dose creating effects on patients.

One of my close relatives had Lung Cancer. It was detected at stage – IV. I asked him not to make the patient go here and there for treatments because nothing can be done. I advised them to follow Dr. Chatterjee's treatment. Patient had neither pain nor any agony till the time he survived.

Yes, Palliative Management of Cancer treatment along with Psorinum Therapy is being quite effective. Dr. Ashim Chatterjee's drug is working on specific cancer patients. The most important attribute of Dr. Chatterjee is that he never accepts defeat. He never returns anyone. Always speaks about trying till the last. He gives the correct advice to both patient and his /her family. This is needed. He always provides appropriate services and advises to patient families at different situations. This can be called the social medicine. This is very important for cancer treatment to day.

The detection of cancer involves three steps – Radiology, Surgery and Pathology. If we go deep into the subject we see that it is pathology that confirms cancer. However, in the past 20 years there has been noticeable improvement in the sphere of Radiology and Radiotherapy with the help of which now radiologists can also determine and confirm cancer to a large extent and advise appropriate future course of treatment. What are your views on this?

Yes, it is true that in the past 20 years Radiology has improved a lot and now we have some tools to determine cancer. But the pathology reports, biopsy slides speak last. We can determine cancer and the part affected by it but what kind of cancer it is determined by the pathologists.

With the improvement in Medical Science, a radiologist can observe that a patient had cancer and now the disease has regressed. But this can be documented either by an oncologist or a general practitioner. But the case has to be FNAC and biopsy proven.

Does that mean to an extent the diagnosis lies with the radiologists?

Yes, of course. As you said that the sphere of Radiology has widened a lot, now-a-days a radiologist's role and responsibility has also expanded.

Sir, you have known Dr. Asim Chatterjee for long 16 years now. On many platforms you have stated that advance cancer patients are doing well under Dr. Chatterjee's treatment and on

many occasions cancer has totally regressed without RT or CT. Here also comes into the picture the role of palliative care that eventually relieves the patient of the tremendous pain he/she is suffering from. You have praised Dr. Chatterjee for running such an institution at his own residence where more stress is given on the palliative aspect of the treatment. You have also come across many cancer patients Dr. Chatterjee has treated over the years who are alive for 3 – 10 years now. You have been a co-author with Dr. Chatterjee on many papers, journals and articles where such cases have been documented. What are your views regarding Dr. Chatterjee's therapy and is the outcome promising enough?

I have never feared to tell the truth. Dr. Chatterjee's treatment has benefitted thousands of cancer patients till now. I have seen patients return from the verge of death. For example, Mr. Sujit Kumar Paul's case, who was a patient from Thakurpukur Cancer Hospital. This case was an FNAC and tissue proven case. Not only this, surgeons at Thakurpukur Cancer Hospital had performed a bypass-surgery and laparotomy on him and were surprised to find how much the cancer had spread from his gall bladder to adjoining organs as well. They also took tissue from the metastatic SOL from liver for several tests. Today, after three years when I review his CT scan plates I see that under Dr. Chatterjee's treatment the patient is doing very well. Cancer has totally regressed and his gall bladder and liver has also turned normal without any scar. His PET scan and CT scan reports are also clear. This is a clinical, radiological and histopathologically proven case. This was an institutional case and post regression Mr. Sujit Kumar Paul was also taken to Thakurpukur Cancer Hospital and there the doctors have given a written acknowledgement to this case. However, he was asked to visit the hospital every six months for medical review.

The source of Dr. Chatterjee's selfless research emerges from the sense of sincere patriotism on the grounds of humanity. I am very hopeful about his work and also about Dr. Aradeep Chatterjee who bears a huge potential to carry on with this work successfully in the near future.

Again Dr. Chatterjee was determined from the very beginning to rebuild the entire structure of cancer treatment. I feel over the years the structure has become more enriched. The module that Dr. Chatterjee has come up with is basically targeted to those 85% of the Indians who come from poor socio-economic background and are not competent enough to bear the huge burden of cancer treatment. Dr. Chatterjee has come up to this position from working among the poor and weak section of our society. Here two methodologies comes into the picture – the development of Science and it's proper utilization in such a way that it would accommodate those people who are lagging behind. This cannot be achieved in one day, one month or one year. This is going to involve years of hard work, dedication, honesty and support of recognised institutions and organizations, and blessings of those people who have been benefitted.

Why is Dr. Chatterjee's Psorinum therapy devoid of any publicity?

Actually he is quite opposed to publicity. He likes to stay away from publicity. Once we

arranged for a Conference at University Institute Hall. But due to too many speakers, all the issues could not be addressed, indeed all present there admitted that Palliative Management Unit at Dr. Chatterjee's residence has been quite beneficial to many.

Conversation with Prof. Anup Majumdar

Today Psorinum Therapy has received recognition on major scientific platforms as an alternative method of treating cancer. It is worth noticing that how come a person coming from a nontechnical background turned out to become a highly technical person facing severe resistances after overcoming critical situations. You have witnessed the rise of Dr. Chatterjee and have been associated with him for a long time. What difference do you see between yesteryear's and today's Dr. Asim Chatterjee?

Well, no such remarkable change that I have come across in Dr. Chatterjee. Previously he used to constantly roam from one place to the other visiting patients in remote areas. Today he doesn't roam but treat patients at his residence-facility. He is the same person – very curious and at the same time very helpful.

It has been 31 years that you know him now. During 1980 – 85 when Dr. Chatterjee was attaining the primary knowledge from Cure Point Nursing Home you were among the three prominent personalities who helped him with all the guidance, Prof. R. N. Brahmachari, Prof. R. S. Bhakta and you. Prof. R. S. Bhakta enlightened him with the basic allopathic treatment and was also associated with Dr. Chatterjee till the time he breathed his last. Although you at that time held a much higher position still you went with Dr. Chatterjee and visited cancer patients at their residences and administered blood transfusion and other supportive treatment. It is very unfortunate that we do not have any pictorial documentation of that time. According to Dr. Chatterjee, "India has never learnt to keep histories". What do you have to say on this?

There are two aspects of this. One is the philosophical and the other is the scientific aspect. Being an oncologist my first priority is relieving the person from the pain he is suffering from. I here admit that the sphere of cancer and its treatment is so vast that 99 per cent of it is unknown to me. After so many years of practising now if a patient approaches me and asks me that would become absolutely cured, I do not have any answer to his question. Suppose two patients visit me who are similar in every aspect, for example, age, gender, height, health, the part of the body affected by cancer etc. I observed one of them getting cured and the other's condition deteriorating continuously, although they are kept on the same medication. Medical science does not have any answer to this kind of extreme behaviour. I sometimes ask myself the question that why is there this gap. Here comes in the picture the scientific aspect. Science has been able to answer a few questions while many other are still unknown. If a patient is alive for, say, two years under my treatment and three years under Dr. Chatterjee's treatment, why is this so? This one year gap is very significant. Also we should keep in mind what quality of life the patient is having in the post-treatment days. Is he actively working or has become bed-ridden. The laboratory diagnosis and tests, the clinical

proof and the effectiveness of the drug also plays a very important role in this regard. Also we should note that how the drug is working and what kind of effect it has on the infected areas. Why a few patients are recovering and while others gradually moving towards their destined ends. Today when a new drug is discovered, we need to look at its side-effects before even considering its positive effect on human body. Chemotherapy has played a very important role in the treatment of cancer till date and it has the potential to impact the standard of living of millions of patients worldwide. Psorinum Therapy has now entered in the main flow of medical and scientific treatment platform and is giving us remarkable results with and without the application of Chemotherapy and Radiotherapy by its side. This non-toxic drug has successfully cured advanced stage lung, liver, gall bladder, stomach and pancreatic cancer.

The inventions of today's modern treatment were done by non-medical personalities by some accident. However, this has benefitted millions of people so far. Psorinum Therapy too from the very beginning of its discovery has benefitted thousands. Over the years the drug has also undergone major developments. I believe that it would do much more justice if it is available to the main stream oncologists in some time in the future after some necessary clinical trials. So far I have seen this drug to be clinically successful and curing, if not curing then extending a patient's life by a few years.

Conversation with Dr. Hiranamoy Mukherjee

Sir, today you are one of the most eminent physician that we have in our country. You have been a subject-matter-expert in Pharmacology and have guided many Ph. D. students till date as well. You have also served as the Head of the Entomology Department in the School of Tropical Medicine. You are at this point of time the only physician who is an MD as well as a Ph. D. You have been long associated with Dr. Chatterjee and always stood by him as a friend, Philosopher and guide. You have time and again acted as a pillar of support for him. Please let us all know how you came to know about Dr. Chatterjee and what brought both of you together?

It was during 1992–92, many people used to visit the School of Tropical Medicine with the objective of working more on the traditional Medicine of cancer and Dr. Chatterjee was one of them. He met with the then Head of the Department of the concerned department. He was time and again asked to experimentally prove the effectiveness of the drug he was working with although he had so many successful case studies to prove his mettle. Dr. Chatterjee returned from there being disappointed. I had somehow noticed him that day. One of my neighbours at that time was suffering from Prostate cancer. One night he fell very critically ill and started bleeding from his mouth and nose. I, somehow, thought of Dr. Chatterjee then and asked the patient's family members to contact him. As soon as the sun rose the patient's family went to Dr. Chatterjee's residence and brought him. Dr. Chatterjee administered the drug on the patient. Soon the patient began sweating and trembling severely. As the day progressed his bleeding stopped too. His pain also ceased considerably. For the next few days the patient continued with the medicine. Gradually his overall health condition improved.

After a few days when medical tests were conducted no trace of cancer was found in his body, only the bones showed a bit of effect left. The patient lived for three months after which he died due to some elderly disease.

I have in due course come across many people who considered Dr. Chatterjee to be a fraud. Some of the patients who were getting treated under Dr. Chatterjee's supervision also stopped taking their medicine once they felt a bit better. Because of this irregularity we never used to contradicting results and this at times used to let us down. Later we thought that first of all we would have to convince people that in order to get cured they would require continuing with the medication throughout their lives. This gave us better result and in many cases cancer regressed completely. It is our misfortune that it is not always possible to give the description of these kinds of case studies.

Dr. Chatterjee for so many years with so much of patience has given his heart and soul in this work for humanity. It is now very important to work more towards the development of the drug. We have wandered many places to perform animal tests during those days. But in many institutions such infrastructure was not available.

In India cancer is spreading at a tremendous rate. Again Psorinum Therapy's primary source is Mite. This is a part of the Entomology stream. In this context what kind of role Psorinum Therapy is expected to play? What are your views regarding the future of this kind of treatment? This has been stated by a few physicians and scientists that this kind of work under the direction of an individual is very rare and has not been witnessed in India at least. What are your views on the entire subject?

This drug is a unique one whose primary source is Mite and the other important component is snake's venom. At the School of Tropical Medicine Dr. Chatterjee used to work on this composition. This drug has been successful so far. But we will be fully assured of its efficacy when this drug would give 100/100 result. Then we will think that yes now we have achieved something.

Our ultimate objective is more and more patient getting benefitted from this initiative. It is very important that this treatment method getting absorbed in the modern management of cancer treatment and cure. The cancer types that Dr. Chatterjee specifically works on are renal-cell carcinoma, different types of carcinoma, osteo-sarcoma and we have witnessed significant results till date. I have seen an advanced stage very young post-surgery cancer patient with non-operable malignant teratoma getting cured. His cancer has totally regressed. He is married now and is leading a healthy life. Many patients like him, from different parts of India and abroad, have come and visited Dr. Chatterjee at his residential hospital facility. People have many expectations from him and I have faith in those people.

In this context I would like to add, as I go back in history we all including Prof. R. N. Brahmachari had asked Dr. Chatterjee time and again to work with the drug's pharmacokinetics and pharmacodynamics which is very essential but he thought otherwise. Presently this work is getting carried on at Chittaranjan National Cancer Institute's Cancer Prevention

Department. Prof. Joydeep Biswas, Director of CNCI, is also very hopeful with the recent developments. Today as I review his work I realise why he did not continue with his research then. Why he did not enter the laboratory leaving aside everything. Today all my questions are getting answered. He was very realistic from the very beginning and the way he handled certain situations others could not even think of. At that time he worked without looking into the drug's pharmaco-kinetics and pharmaco-dynamics and gave this treatment method a concrete ground. At CNCI the phase-I of research is almost complete and soon the phase-II would come into effect.

You have never charged any fees for the service you have rendered at CCMRCC. Dr. Chatterjee has appreciated this gesture of yours so many times. What was the reason behind this?

This scientific work has always been directed to serve the humanity. I feel honoured to be associated with Dr. Chatterjee's mission. I am very hopeful that in future Psorinum Therapy would open new avenues for the treatment of cancer and eventually wipe it off. Again Dr. Chatterjee has been more than a friend to me. We share a unique bond and I have always shown excitement whenever he thought of taking this a step further and I will continue supporting him in the future as well.

Conversation with Prof. Amiyo Kumar Hati

Sir, you were the former Director of the School of Tropical Medicine and also associated with the Directorate of Science and Technology. In the 1990s the scientific community was very rigid in their thoughts and beliefs. During those times you supported Dr. Chatterjee and gave him the opportunity to work in the School of Tropical Medicine under your supervision. Further, under the banner of the School of Tropical Medicine his paper titled 'Treatment of Cancer – A Step Forward' was presented in Rajasthan Session of Science Congress and his paper is also included in the twelve years work of the School of Tropical Medicine. What led you to go out of the way to support Dr. Chatterjee?

Dr. Asim Chatterjee came to the School of Tropical Medicine with a request that whether we would be able to help him in his research on Psorinum therapy. We were always very open-minded and encouraged any new work. We tried to help him in his research. We gave him the opportunity to study in the School of Tropical Medicine itself. We get Psorinum from *Sarcoptes scabiei* which is a kind of mite. It is very easily available in the School of Tropical Medicine than anywhere else. However, it has not materialized yet but efforts are going on to materialize the culture of Scabies mite. We were able to assist Dr. Chatterjee in every way possible in his research. Later on we presented his paper in the Rajasthan Session of Science Congress under the banner of the School of Tropical Medicine. At that time I was the Head of Department of the Entomology as well as the Director of the School of Tropical Medicine. I was also the member of the Program Advisory Committee of the Directorate of Science and Technology.

I am glad that his work has reached the global platform today and many articles and

papers have been published in many international journals like Lancet, Journal of Thoracic Oncology, Journal of Clinical Oncology and many more. I would say that we all should have an open-mind and accept and encourage any new approach. The work that Dr. Chatterjee is doing is really inspiring. Dr. Chatterjee and his team of doctors are working on the diagnosis of the disease, its treatment, the most important, rehabilitation as well as terminal care of the patients. They are keeping a close eye on all the aspects of cancer. Dr. Chatterjee is also trying to create awareness among the general masses regarding cancer and I whole-heartedly appreciate it. My best wishes are always with him and I further wish him more and more success in near future.



Dr. Asim Chatterjee with Prof. Amiyo Kumar Hati

FROM THE PERSPECTIVE OF A YOUNG PHYSICIAN

Dr. Sudipto Chatterjee

Dr. Sudipto Chatterjee is actively engaged in promoting human welfare by saving lives, relieve suffering, and maintaining human dignity. He is currently associated with us working at Critical Cancer Management Research Centre and Clinic (CCMRCC), and provides service to the socio-economically backward people and terminally-ill patients who have been turned down by major institutions through his supportive treatment. In the following paper he has put forward his thoughts beautifully in a few words.

CURRENT SCENARIO OF CANCER: SCIENCE AND SOCIETY

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(I) Cancer: Present Scenario

Cancers are a large family of diseases that involve abnormal cell growth with the potential to invade or spread to other parts of the body.[1][2] They form a subset of neoplasms. A neoplasm or tumor is a group of cells that have undergone unregulated growth and will often form a mass or lump, but may be distributed diffusely.[3][4]

All tumor cells show the six hallmarks of cancer. These characteristics are required to produce a malignant tumor. They include:[5]

- Cell growth and division absent the proper signals
- Continuous growth and division even given contrary signals
- Avoidance of programmed cell death
- Limitless number of cell divisions
- Promoting blood vessel construction
- Invasion of tissue and formation of metastases[6]

The progression from normal cells to cells that can form a detectable mass to outright cancer involves multiple steps known as malignant progression.[7]

The majority of cancers, some 90–95% of cases, are due to environmental factors. The remaining 5–10% are due to inherited genetics.[8] Environmental, as used by cancer researchers, means any cause that is not inherited genetically, such as lifestyle, economic and behavioural factors and not merely pollution.[9] Common environmental factors that contribute to cancer death include tobacco (25–30%), diet and obesity (30–35%), infections (15–20%), radiation (both ionizing and non-ionizing, up to 10%), stress, lack of physical activity and environmental pollutants.

It is not generally possible to prove what caused a particular cancer, because the various causes do not have specific fingerprints. For example, if a person who uses tobacco heavily

develops lung cancer, then it was probably caused by the tobacco use, but since everyone has a small chance of developing lung cancer as a result of air pollution or radiation, the cancer may have developed for one of those reasons. Excepting the rare transmissions that occur with pregnancies and occasional organ donors, cancer is generally not a transmissible disease.[10]

Exposure to particular substances have been linked to specific types of cancer. These substances are called carcinogens. Tobacco smoke, for example, causes 90% of lung cancer. [11] It also causes cancer in the larynx, head, neck, stomach, bladder, kidney, esophagus and pancreas.[12]

Tobacco smoke contains over fifty known carcinogens, including nitrosamines and polycyclic aromatic hydrocarbons.[13] Diet, physical inactivity and obesity are related to up to 30–35% of cancer deaths. In the United States excess body weight is associated with the development of many types of cancer and is a factor in 14–20% of cancer deaths.[14] Worldwide approximately 18% of cancer deaths are related to infectious diseases. This proportion ranges from a high of 25% in Africa to less than 10% in the developed world.[15] Viruses are the usual infectious agents that cause cancer but cancer bacteria and parasites may also play a role. Up to 10% of invasive cancers are related to radiation exposure, including both ionizing radiation and nonionizing ultraviolet radiation.[16] Additionally, the majority of non-invasive cancers are non-melanoma skin cancers caused by non-ionizing ultraviolet radiation. The vast majority of cancers are non-hereditary (“sporadic”). Hereditary cancers are primarily caused by an inherited genetic defect. Less than 0.3% of the population are carriers of a genetic mutation that has a large effect on cancer risk and these cause less than 3–10% of cancer.[17] Some hormones play a role in the development of cancer by promoting cell proliferation.[18] Insulin-like growth factors and their binding proteins play a key role in cancer cell proliferation, differentiation and apoptosis, suggesting possible involvement in carcinogenesis.[19]

In 2008, approximately 12.7 million cancers were diagnosed (excluding non-melanoma skin cancers and other non-invasive cancers) and in 2010 nearly 7.98 million people died.[20] Cancers account for approximately 13% of deaths. The most common are lung cancer (1.4 million deaths), stomach cancer (740,000), liver cancer (700,000), colorectal cancer(610,000) and breast cancer (460,000).[21] This makes invasive cancer the leading cause of death in the developed worldand the second leading in the developing world. Over half of cases occur in the developing world.

Deaths from cancer were 5.8 million in 1990. Deaths have been increasing primarily due to longer lifespans and lifestyle changes in the developing world. The most significant risk factor for developing cancer is age.[22] Although it is possible for cancer to strike at any age, most patients with invasive cancer are over 65. According to cancer researcher Robert A. Weinberg, “If we lived long enough, sooner or later we all would get cancer.”[23] Some of the association between aging and cancer is attributed to immunosenescence, errors accumulated in DNA over a lifetime[24] and age-related changes in the endocrine system.[25] Aging’s

effect on cancer is complicated by factors such as DNA damage and inflammation promoting it and factors such as vascular aging and endocrine changes inhibiting it.[26]

(II) Cancer: History, Society and Culture

Cancer has existed for all of human history.[27] The earliest written record regarding cancer is from circa 1600 BC in the Egyptian Edwin Smith Papyrus and describes breast cancer. Hippocrates (ca. 460 BC – ca. 370 BC) described several kinds of cancer, referring to them with the Greek word *καρκίνοϛ* *karkinos* (crab or crayfish). This name comes from the appearance of the cut surface of a solid malignant tumor, with “the veins stretched on all sides as the animal the crab has its feet, whence it derives its name”.[28] Galen stated that “cancer of the breast is so called because of the fancied resemblance to a crab given by the lateral prolongations of the tumor and the adjacent distended veins”.[29] Celsus (ca. 25 BC – 50 AD) translated *karkinos* into the Latin *cancer*, also meaning crab and recommended surgery as treatment. Galen (2nd century AD) disagreed with the use of surgery and recommended purgatives instead. These recommendations largely stood for 1000 years.

In the 15th, 16th and 17th centuries, it became acceptable for doctors to dissect bodies to discover the cause of death.[30] The German professor Wilhelm Fabry believed that breast cancer was caused by a milk clot in a mammary duct. The Dutch professor Francois de la Boe Sylvius, a follower of Descartes, believed that all disease was the outcome of chemical processes and that acidic lymph fluid was the cause of cancer. His contemporary Nicolaes Tulp believed that cancer was a poison that slowly spreads and concluded that it was contagious.[31]

The physician John Hill described tobacco snuff as the cause of nose cancer in 1761. This was followed by the report in 1775 by British surgeon Percivall Pott that chimney sweeps’ carcinoma, a cancer of the scrotum, was a common disease among chimney sweeps.[32] With the widespread use of the microscope in the 18th century, it was discovered that the ‘cancer poison’ spread from the primary tumor through the lymph nodes to other sites (“metastasis”) This view of the disease was first formulated by the English surgeon Campbell De Morgan between 1871 and 1874.[33]

Though many diseases (such as heart failure) may have a worse prognosis than most cases of cancer, cancer is the subject of widespread fear and taboos. The euphemism “after a long illness” is still commonly used (2012), reflecting an apparent stigma.[34] This deep belief that cancer is necessarily a difficult and usually deadly disease is reflected in the systems chosen by society to compile cancer statistics: the most common form of cancer—non-melanoma skin cancers, accounting for about one-third of cancer cases worldwide, but very few deaths[35] [36] are excluded from cancer statistics specifically because they are easily treated and almost always cured, often in a single, short, outpatient procedure.[37]

Cancer is regarded as a disease that must be “fought” to end the “civil insurrection”; a War on Cancer was declared in the US. Military metaphors are particularly common in descriptions of cancer’s human effects and they emphasize both the state of the patient’s health

and the need to take immediate, decisive actions himself, rather than to delay, to ignore, or to rely entirely on others. The military metaphors also help rationalize radical, destructive treatments.[38][39]

In the 1970s, a relatively popular alternative cancer treatment in the US was a specialized form of talk therapy, based on the idea that cancer was caused by a bad attitude.[40] People with a “cancer personality”—depressed, repressed, self-loathing and afraid to express their emotions—were believed to have manifested cancer through subconscious desire. Some psychotherapists said that treatment to change the patient’s outlook on life would cure the cancer. Among other effects, this belief allowed society to blame the victim for having caused the cancer (by “wanting” it) or having prevented its cure (by not becoming a sufficiently happy, fearless and loving person).[41] It also increased patients’ anxiety, as they incorrectly believed that natural emotions of sadness, anger or fear shorten their lives. The idea was ridiculed by Susan Sontag, who published *Illness as Metaphor* while recovering from treatment for breast cancer in 1978. Although the original idea is now generally regarded as nonsense, the idea partly persists in a reduced form with a widespread, but incorrect, belief that deliberately cultivating a habit of positive thinking will increase survival. This notion is particularly strong in breast cancer culture.

One idea about why people with cancer are blamed or stigmatized, called the just-world hypothesis, is that blaming cancer on the patient’s actions or attitudes allows the blamers to regain a sense of control. This is based upon the blamers’ belief that the world is fundamentally just and so any dangerous illness, like cancer, must be a type of punishment for bad choices, because in a just world, bad things would not happen to good people.[42] In 2007, the overall costs of cancer in the US—including treatment and indirect mortality expenses (such as lost productivity in the workplace) — was estimated to be \$226.8 billion. In 2009, 32% of Hispanics and 10% of children 17 years old or younger lacked health insurance; “uninsured patients and those from ethnic minorities are substantially more likely to be diagnosed with cancer at a later stage, when treatment can be more extensive and more costly.”[43]

(III) Current Research: Role of Psorinum Therapy

Because cancer is a class of diseases,[44][45] it is unlikely that there will ever be a single “cure for cancer” any more than there will be a single treatment for all infectious diseases. [46] Angiogenesis inhibitors were once incorrectly thought to have potential as a “silver bullet” treatment applicable to many types of cancer.[47] Angiogenesis inhibitors and other cancer therapeutics are used in combination to reduce cancer morbidity and mortality. [48] Experimental cancer treatments are studied in clinical trials to compare the proposed treatment to the best existing treatment. Treatments that succeeded in one cancer type can be tested against other types.[49] Diagnostic tests are under development to better target the right therapies to the right patients, based on their individual biology.[50]

Cancer research focuses on the following issues:

- Agents (e.g. viruses) and events (e.g. mutations) that cause or facilitate genetic changes in cells destined to become cancer.
- The precise nature of the genetic damage and the genes that are affected by it.
- The consequences of those genetic changes on the biology of the cell, both in generating the defining properties of a cancer cell and in facilitating additional genetic events that lead to further progression of the cancer.

The improved understanding of molecular biology and cellular biology due to cancer research has led to new treatments for cancer since US President Richard Nixon declared the “War on Cancer” in 1971. Since then, the country has spent over \$200 billion on cancer research, including resources from public and private sectors.[51] The cancer death rate (adjusting for size and age of the population) declined by five percent between 1950 and 2005.[52]

Competition for financial resources appears to have suppressed the creativity, cooperation, risk-taking and original thinking required to make fundamental discoveries, unduly favoring low-risk research into small incremental advancements over riskier, more innovative research. Other consequences of competition appear to be many studies with dramatic claims whose results cannot be replicated and perverse incentives that encourage grantee institutions to grow without making sufficient investments in their own faculty and facilities.[53][54][55].

In this context, Psorinum Therapy has developed a new horizon in the treatment of Cancer. It is mainly concentrated in treatment of Lung, Stomach, Gall Bladder, Pancreas, Liver and Kidney tumors. Many studies were published in different journals all over the world like, Journal of Thoracic Oncology, Journal of Clinical Oncology, The Lancet, Oral Oncology, Tobacco Counters Health etc. These studies have showed that how Psorinum works effectively in terminally ill cancer patients. Most of these are resistant and relapsed cases. It is very much surprising that in many cases there are tumour regression. In many cases spread of metastatic tumours have been arrested and patients can carry a near normal daily life. The drug can be taken easily orally and treatment can be continued at outdoor basis. Few patients who are critically ill are admitted in hospital for supportive medical care in some of which Psorinum can be given intravenously by mixing with dextrose solution. The main advantages of the drug are, it is non-toxic and cheap. Patient can continue the drug easily as home based therapy. In Kolkata the treatment is going at Critical Care Management Research Centre and Clinic, 381, S. K. Deb Road, Kolkata – 700 048. The research with the molecule was started in 1983. From that time Dr. Ashim Chatterjee carried out different studies with this drug in Indian Cultivation of Science, Tata Memorial Cancer Hospital, Chittaranjan Cancer Hospital etc. Many patients are still taking the drug for different types of cancer. This proves that the drug is time tested.

From my experience I am giving two case studies in which I was also involved from the part of treatment of internal medicine.

- 1) Mrs. Krishna Banerjee (70 years old) was admitted with huge pelvic mass involving uterine wall, bladder dome, sigmoid colon, left lateral pelvic wall with cystic peritoneal deposits. Patient was referred from Tata Medical Centre and they refused to do any treatment just after taking a biopsy. The patient was treated with Psorinum and other supportive medical management. Now the tumour is much small in size and patient is carrying normal daily life. Supportive documents are attached at the end of this paper.

Reports and supporting documents of Mrs. Krishna Banerjee



Patient Name	: Krishna Banerjee	Ref. Dr.	: Dr. J. Sharma
Age	: 70 Years	Date of Test	: 13.06.2016
Sex	: Female	Date of Report	: 13.06.2016
		ID No	: 59936

CECT SCAN OF WHOLE ABDOMEN

Contrast enhanced CT scan of whole abdomen was done taking 5 mm contiguous axial section with sagittal and coronal reformatt images.

SCANOGRAM : Supine frontal projection of abdomen is non-contributory.

REPORT:

Lower Pleural Space :- Mild pleural thickening in both side.

Liver :-

Liver is mildly enlarged. Otherwise, it is normal in outline & enhancement. Generalised decrease in parenchymal attenuation. No focal lesion is seen. Intrahepatic tubular structures are normal in course & caliber. Porta hepatis is normal.

Gall Bladder:-

Gall bladder is visualised. No mass is seen. No radio-opaque calculi seen within gall bladder lumen [Radio – lucent calculi may be missed by CT scan].

Common Bile Duct :-

Common duct is not dilated nor any radiodense calculus in it.

Pancreas :-

It is normal in size, outline, density & enhancement. No focal lesion is seen. Main pancreatic duct is not dilated. No calcification is seen.

Spleen :-

It is normal in size, outline, density & enhancement. No focal lesion is seen.

Kidneys :-

Both are normal in size, outline, density & enhancement. Pelvicalyceal system is dilated on the left side & excretion is seen in both systems. No calculous or focal lesion is seen.

Adrenals :-

All the three limbs of adrenal glands are well outlined. There is no focal thickening in any of its limb.

Ureters :-

Right – Right ureter is normal in course, caliber.

Left – Left ureter is mildly dilated upto its lower third where it is seen to be compressed by the SOL described below.

Urinary Bladder :-

It is normal in capacity and wall thickness. No intraluminal or mural pathology is seen. Indentation over the left lateral wall of urinary bladder by the SOL described below is seen.

Uterus:-

Not visualised.

The SOL:-

A large (measuring 15.66 cm X 12.50 cm X 11.57 cm) thick walled, marginally enhancing, cystic SOL containing high density fluid, is seen arising from the pelvis on the left side & extending just superior to the pelvic brim. The mass is seen indenting the left lateral wall of urinary bladder. The mass is also seen to cause compression over the lower third of left ureter. Displacement of adjacent loops of gut seen.

Bowel & Mesentery :-

No obvious bowel wall thickening or mass is seen. Mesenteric density is normal. No obvious mesenteric lymphadenopathy is seen. No free fluid is seen in abdomen.

Retroperitoneum :-


IVC & aorta are normal in course, caliber. No upper retroperitoneal lymphadenopathy is seen.

Bones & Soft tissue :-

Bones & soft tissues are of normal density & enhancement.

IMPRESSION: CECT scan of whole abdomen reveals –

- A large, thick walled, marginally enhancing, cystic SOL containing high density fluid arising from the pelvis on the left side & extending just superior to the pelvic brim & causing extensive compression over the lower third of left ureter with proximal hydronephrosis and mass effect over adjacent structure as detailed above – suggestive of ovarian neoplastic.
- Mild hepatomegaly with generalised fatty infiltration of liver.
- Mild bilateral pleural thickening.


Dr. Nilanjana Datta Chaudhuri
MD (Radio – Diagnosis)
Consultant Radiologist

Dr. S. L. Kedia
(MD, PGI – Chandigarh)
Consultant Radiologist

Dr. M. Roy Chowdhury (Senior)
MBBS, MD
Consultant Radiologist



TATA MEDICAL CENTER

2

Tata Medical Center

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Website: www.tmckolkata.com

Patient Evaluation Summary

MR No. : MR/16/007248 Name : Mrs. KRISHNA BANERJEE
Patient No. : OP/16/018499 Sex : FEMALE Age : 70 Y 0 M
Address : 51/7 ANJAN GARH, BIRATI PS- AIRPORT, 24 PARGANAS NORTH, WEST BENGAL-700051, INDIA
Assessment Dt : 21/06/2016 16:15:47

VITAL PARAMETERS (Last Measured)

Height: 150 cms Weight: 52.8 kg BSA: 1.48 sq m BMI: 23.47 kg/sq m
Pulse: 116 /min Temperature: 98.9 DegF BP: 150 / 60 mmHg Respiratory Rate: N/A

CHIEF COMPLAINTS

PROGRESSIVE WEAKNESS FOR PAST 2 WEEKS. RECENTLY DETECTED WITH PELVIC MASS WITH SEVERE ANAEMIA. MANAGED AT LOCAL HOSPITAL. RECEIVED BLOOD TRANSFUSION 3 UNITS.
NO DEFINITE LATERALISED WEAKNESS OF ANY LIMBS. NO LOC/ CONVULSIONS. MILD SLURRING OF SPEECH WITH DEVIATION OF FACE TO RT.

ON EXAMINATION

CNS - HMF - CONSCIOUS, ALERT, ORIENTED. CN - 7UMN TYPE LEFT VII N PARESIS. NO OTHER CN IMPAIRMENT.
MOTOR - NORMAL TONE, POWER - GRADE 5/5 IN ALL FOUR LIMBS, DTR - NORMAL. PLANTAR - B/L FLEXOR.
SENSORY - NAD. ROMBERG'S - NEGATIVE
CEREBELLAR - NAD.
CHEST - VBS
CVS - S1, S2.

INVESTIGATION

Investigation

ELEC/RENAL PANEL (Uro Creatinine, Na, K)
GLUCOSE AC PL (Pst)
HbA1c - GLYCOSYLATED Hb
THYROID PANEL I - (T3, T4 & TSH)
CBC+DIFF
MRI BRAIN

ADVICE

REVIEW WITH REPORTS.

(22/6/16)

Remarks

Dr. Anirban Laha
MBBS, DNB General Medicine
Fellow, Regn. No. : MCI/31228
Department of General Medicine

-End of Report-

Please take an appointment for next visit. Ph : 033-6605 7222, Email id : appointment@tmckolkata.com

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Tata Medical Center

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Department of Histopathology

Run Date: 16/08/2016 12:00

MR No. : MR/16/007248 Request No. : SQ/16/279090
Name : Mrs. Krishna Banerjee Patient ID : IP/16/004624
Age : 70 Y 1 M Sex : Female
Age at time of Sample Collection : 70 Y 1 M Patient Location : SURBAY/SURBY2/SUR 12
Reported on : 09/08/2016 09:07:34
Referring Doctor : Jaydip Bhaumik

Lab. Ref. No. : HT/16/005189

Specimen : TISSUE (SE/16/116551)

Collected On : 02/08/2016 07:50:05 Received On : 02/08/2016 09:05

SMALL SPECIMEN

SPECIMEN

Omentum - excision biopsy.

DIAGNOSIS

INVOLVED BY SEROUS CARCINOMA.

Please see comment.

COMMENTS

Section shows papillae and clusters of uniform population of cells with minimal nuclear pleomorphism and small prominent nucleoli. Numerous psammoma bodies are seen. Immunohistochemistry done at TMC shows the tumour cells to be strong and diffuse positive for WT-1, PAX-8 and p53 (mutant type); supporting the above diagnosis and an ovarian/tubal/peritoneal primary. Morphologically tumour falls into the category of LOW GRADE SEROUS CARCINOMA. However; in view of strong and diffuse p53 expression, high grade serous needs to be ruled out. Suggest biopsy from the pelvic mass for a definite sub-categorisation. (KG/GM)

GROSS DESCRIPTION

A. Received in formalin labelled with patient name, MR No. and "Omentum" is a fibrofatty tissue measuring 13.5 x 3.0 x 0.7 cm. Externally no deposit identified. Representative section submitted. Grossed by Dr.Kavita Gupta.

LIST OF SECTIONS

Cassette A/1 - A/8 : Omentum, RSS.

-: End of Report :-

SLIDES AND PARAFFIN BLOCKS OF TISSUES PROCESSED AT TMC KOLKATA WILL BE STORED FOR 20 YEARS.
TISSUE SPECIMEN IN TMC KOLKATA WILL BE DISCARDED 3 MONTHS AFTER FINAL REPORT.

Authorised By
Dr. Geetashree Mukherjee

"This report is electronically generated"
"Doesn't relate only to the items tested"



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Patient Evaluation Summary

MR No. : MR/16/007248 Name : Mrs. KRISHNA BANERJEE
Patient No. : OP/16/018779 Sex : FEMALE Age : 70 Y O M
Address : 51/7 ANJAN GARH, BIRATI PS- AIRPORT, 24 PARGANAS NORTH, WEST BENGAL-700051, INDIA
Assessment Dt : 28/06/2016 15:11:56

VITAL PARAMETERS (Last Measured)

Height: 150 cms Weight: N/A BSA: N/A BMI: N/A
Pulse: N/A Temperature: N/A BP: N/A Respiratory Rate: N/A

CHIEF COMPLAINTS

Patient has come for discussion regarding further management

HISTORY

H/o sudden onset weakness 2 weeks back. Loss of bladder and bowel control.
History of treatment in the current illness: Blood transfusion - 3 units. Routine blood investigations were done and was found to have Severe anaemia. CT abdomen was done on finding abdominal mass.
Menstrual history: post menopausal.
Obstetric history: P1
Past Medical history: No comorbidities.
Past Surgical history: Nil

CT s/o large cystic mass in left ovary. Thick walled cyst. Left hydronephrosis.

ON EXAMINATION

Not examined today

REFERRALS

Anaesthesia
General Medicine
Suddhasatwya Chatterjee

DISCUSSION

✓ Seen with Dr Jaydip Bhaumik
As the tumour markers are only mildly raised, malignant neoplasm is less likely. Laparotomy can be undertaken after patient is declared fit from anaesthesia point of view.

ADVICE

To see anaesthesia after clearance from medicine.

Next Followup Date : 07/07/2016

Radhakanta Pal
Dr. Radhakanta Pal
MBBS, MS (Gynae & Obs)
Fellow, Regn. No. : 53704
Department of Gynaecological
Surgery

Please take an appointment for next visit. Ph : 033-6605 7222, Email id : appointment@tmckolkata.com

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Patient Evaluation Summary

MR No. : MR/16/007248 Name : Mrs. KRISHNA BANERJEE
Patient No. : OP/16/018779 Sex : FEMALE Age : 70 Y 0 M
Address : 51/7 ANJAN GARH, BIRATI PS- AIRPORT, 24 PARGANAS NORTH, WEST BENGAL-700051, INDIA
Assessment Dt : 04/07/2016 18:17:47

VITAL PARAMETERS (Last Measured)

Height: 150 cms Weight: 48 kg BSA: 1.41 sq m BMI: 21.33 kg/sq m
Pulse: 108 /min Temperature: 96.7 DegF BP: 149 / 77 mmHg Respiratory Rate: 18/ min

CHIEF COMPLAINTS

PROGRESSIVE WEAKNESS FOR PAST 2 WEEKS, RECENTLY DETECTED WITH PELVIC MASS WITH SEVERE ANAEMIA. MANAGED AT LOCAL HOSPITAL. RECEIVED BLOOD TRANSFUSION 3 UNITS.
NO DEFINITE LATERALISED WEAKNESS OF ANY LIMBS. NO LOC/ CONVULSIONS. MILD SLURRING OF SPEECH WITH DEVIATION OF FACE TO RT.

ON EXAMINATION

CNS - HMF - CONSCIOUS, ALERT, ORIENTED. CN - 3UMN TYPE LEFT VII N PARESIS. NO OTHER CN IMPAIRMENT.
MOTOR - NORMAL TONE, POWER - GRADE 5/5 IN ALL FOUR LIMBS, DTR - NORMAL. PLANTAR - B/L FLEXOR.
SENSORY - NAD. ROMBERG'S - NEGATIVE
CEREBELLAR - NAD.
CHEST - VBS, B/L (R>L) CREPTS (INSPIRATORY) . NO RHONCHI
CVS - S1, S2.
CXR - PROMINENCE OF RT. B/L HILAR SHADOWS.

INVESTIGATION RESULTS

(* Indicates Provisional Report)

BLOOD CULTURE AEROBIC : Negative

SENSITIVITY

(23/06/2016

13:42:46)

NA/K (28/06/2016

13:55:45)

SODIUM : 142 , POTASSIUM : 5.6

MEDICATION

1. Tab THYRONORM 50MCG TAB(1) (1.00 TABLET) X 30 Days (Once a day) from 04/07/2016
2. Tab NEUROBION - FORTE TABS (E MERCK)(1) (1.00 TABLET) X 30 Days (Twice a day- 8:00AM and 8:00PM) from 04/07/2016
3. Tab SELOKEN XL 25MG TAB(1) (1.00 TABLET) X 30 Days (Once a day) from 04/07/2016

DISCUSSION

PRELIMINARY REPORTS REVEAL: HYPOTHYROIDISM (TSH 8.9); HYPOKALEMIA (K- 2.6); ANAEMIA (NORMOCYTIC, NORMOCHROMIC)-HB- (6.8), TLC - 23040 (N- 86%).
MRI- BRAIN - SCATTERED DISCRETE ALTERED SIGNAL INTENSITIES IN B/L PERIVENTRICULAR CORONA RADIATA, CENTRUM SEMIOVALE- LEUKARIOISIS, B/L SUBDURAL HEMORRHAGE, ALTERED SIGNAL INTENSITY IN TECTUM OF MID BRAIN, DORSAL PONS ? CAUSE. EVIDENCE OF CORTICAL ATROPHY.

ADVICE

ADMIT ONE DAY BEFORE SURGERY FOR BP AND VITALS MONITORING

BCa - NSR, RBBB
Bcho - LvtH, arthc Slenon
pmi Calcified

Dr. Suddhasatwya Chatterjee
Consultant
Department of General Medicine

Please take an appointment for next visit. Ph : 033-6605 7222, Email id : appointment@tmckolkata.com

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Run Date : 03/08/2016 10:15:24

Discharge Summary

MR No. : MR/16/007248 Name : Mrs. KRISHNA BANERJEE
Patient No. : IP/16/004624 Sex : Female Age : 70 Y 1 M
Address : 51/7 ANJAN GARH, BIRATI PS- AIRPORT, 24 PARGANAS NORTH, WEST BENGAL-700051, INDIA
Admitting Department : Gynaecological Surgery Admitting Doctor : Jaydip Bhaumik
Admission Date : 30/07/2016 19:07:52 Discharge Date :

DIAGNOSIS

Pelvic SOL

HISTORY

70 year old lady with recent onset generalised weakness with severe anaemia diagnosed to have left ovarian cyst with left hydronephrosis.
Clinical Impression: ? Mucinous cyst of ovary with ?? CVA.
CA125: 71; CEA/CA19-9: WNL

ON EXAMINATION

Vitals stable
no pallor/pedal edema
P/A: soft, wound healthy

DISCUSSION/PROGRESS IN WARD

Exploratory laparotomy on 1/8/16 : A large fixed mass arising from sigmoid colon involving the mesentery and retroperitoneum. Omentum adherent to the mass and bladder. Omental biopsy taken as it was not possible to take biopsy from mass without injuring sigmoid colon.
Postop period uneventful.

OT NOTES

Date: 01/08/2016
Procedures: Exploratory Laparotomy
Reconstruction: No
Reconstruction Notes:

Surgeon notes: Surgery : Exploratory Laparotomy Proceed
Indication : Pelvic mass
Surgeons :
1. Dr. Jaydip Bhaumik
2. Dr. Rupashree Dasgupta

Findings

1. No ascites
2. Large pelvic mass involving the sigmoid colon. Uterus and ovaries not seen separately.
3. Omentum adherent to bladder and sigmoid colon.
4. Pelvis completely obliterated with the mass.

Procedure:

Abdomen opened in layers through midline supra umbilical incision.
Omental adhesion divided, and omentum sent for Histopathology.

Decision was taken to abandon the surgery for the following reasons.

The patient would have needed extensive surgery including anterior exenteration, bowel resection and anastomosis, which was not possible based on the patient's poor performance status and antecedent comorbidities.

RECOMMENDATIONS

- Normal diet
- Daily shower with soap and water
- Keep the wound/port sites dry and clean.
- Tab Pyrilgesic 650 mg thrice a day for 5 days and then SOS for pain
- Tab Rantec 150 mg twice a day for 5 days
- TED stockings to be used in both lower limbs for 6 weeks
- Incentive spirometry @ 800ml 20 times every 2 hourly.
- To continue all chronic medications as before.
- To follow up in Gynae OPD on 16/08/16 for suture evaluation and final HPE.
- To take appointment with Medical/Radiation oncology before discharge.
- To attend ER sos or call at 033-66057247 (9am-6pm) for any queries or contact customer care in case of emergencies beyond working hours

Dr. Swati Mittal
Fellow, Regn. No. : 12-43710
Department of Gynaecological
Surgery

-End of Report -

16/08/16
AS

Date: 16/08/16

Quadra Medical Services Pvt. Ltd.

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Pg.# 1 of 2

V.Id. : 16I26/190

Patient Name : Ms. Krishna Banerjee

Age : 70years , Sex : Female

Referred by : Dr. Ashim Chatterjee..

Test Date : 26/09/16

Report Date : 26/09/16

CT SCAN OF WHOLE ABDOMEN

Plain and contrast enhanced CT scan of whole abdomen done with oral and rectal contrast using a 128 slice spiral scanner

LIVER

Liver is normal in size, outline and density. A tiny cyst (5 mm) is seen in segment VIII of liver. No other focal parenchymal lesion is seen. Intrahepatic tubular structures are normal. Porta hepatis is seen normal. No abnormality is seen in the gall bladder fossa.

SPLEEN

Spleen is normal in size, shape, outline and density. No focal lesion is seen. The splenoportal axis is patent and is normal in dimensions.

PANCREAS

Pancreas is normal in size, shape, outline and density. No focal parenchymal lesion is seen. The pancreatic duct is not dilated. No peripancreatic lesion is present.

BOTH ADRENALS

Both adrenals are not enlarged.

RIGHT KIDNEY AND RIGHT URETER

Right kidney is normal in position, lie, size, outline, density and excretion pattern. Small simple cortical cyst is seen in kidney. The pelvicalyceal system is normal. No calculus or hydronephrosis is seen. Right ureter is not dilated.

LEFT KIDNEY AND LEFT URETER

Left kidney is normal in position, lie, size, outline, density and excretion pattern. Small simple cortical cyst is seen in kidney. Hydronephrosis is seen in left kidney with dilated pelvis and narrowing at pelvi-ureteric junction. The ureter at pelvis is abutted and compressed by pelvic mass. No calculus is seen.

PERITONEUM AND RETROPERITONEUM

Aorta and IVC are seen normal. No para-aortic or retroperitoneal lymphadenopathy is seen. No ascites is seen. There is a cystic space occupying area of 34 x 35 mm size in relation with wall of left sided colon – likely peritoneal deposit.

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Pg.# 2 of 2

V.Id. : 16I26/190

Patient Name : Ms. Krishna Banerjee

Age : 70years, Sex : Female

Referred by : Dr. Ashim Chatterjee..

Test Date : 26/09/16

Report Date : 26/09/16

URINARY BLADDER, PELVIS AND BOWEL

There is a heterogenous irregular thick walled cystic space occupying lesion measuring approximately 67 x 51 x 57 mms size in pelvis just to the left side of midline inseparable from uterine wall, left lateral pelvic wall with vascular encasement and having loss of fat plane with sigmoid colon having thickening of wall. The space occupying lesion is also abutting bladder dome, with loss of fat plane at places.

Rest of the bowel loops are normal.

Ovaries are not separately seen.

No enlarged pelvic nodes are seen.

WALL

Degenerative changes are seen in the vertebrae.

Grade I spondylolisthesis of L5 over S1 with spondylolysis is seen.

Parietal and para vertebral soft tissues are normal.

IMPRESSION:

Thick wall cystic pelvic mass involving uterine wall, bladder dome, sigmoid colon, left lateral pelvic wall with cystic peritoneal deposits.

In comparison with previous CT of 13.06.2016, the size of the space occupying lesion has reduced.


DR. SNEHASIS SARKAR, MD
CONSULTANT RADIOLOGIST

DR. PINAK PANI BHATTACHARYA
MD(PGIMER)DNB
CONSULTANT RADIOLOGIST

DR. MANOJ JAIN, MD
CONSULTANT RADIOLOGIST

DR. SUNETRA MUKHERJEE
MD RADIOLOGY
CONSULTANT RADIOLOGIST

DR. CHATURBHUI LAL RAJAK
MD(PGIMER)DNB
CONSULTANT RADIOLOGIST

DR. BHASWATI MUKHERJEE
DMRD, MD
CONSULTANT RADIOLOGIST

DR. DEEPAK B.
MD, DNB, DMRD
CONSULTANT RADIOLOGIST

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Pg.# 1 of 1

V.Id. : 16I26/190
Patient Name : Ms. Krishna Banerjee
Age : 70years , Sex : Female
Referred by : Dr. Ashim Chatterjee..

Test Date : 26/09/16

Report Date : 26/09/16

X-RAY REPORT OF CHEST PA VIEW

Bony cage and soft tissues are normal.

Domes of diaphragm and costophrenic angles are normal.

The mediastinum is central. The cardiac silhouette is normal in size and configuration.

The hila are normal in size, shape and outline.

The lung fields are well expanded and clear. No active parenchymal lesion is seen.

IMPRESSION:

Study is within normal limits.



DR. SUNETRA MUKHERJEE
MD RADIOLOGY
CONSULTANT RADIOLOGIST

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CONSULTANT RADIOLOGIST

DR. MANOJ JAIN, MD
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DR. CHATURBHUI LAL RAJAK
CONSULTANT RADIOLOGIST

DR. BHASWATI MUKHERJEE
CONSULTANT RADIOLOGIST

DR. DEEPAK P.
CONSULTANT RADIOLOGIST



Immunopath diagnostic centre

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Kolkata - 700 074 • Phone : 033-2548-3000
E-mail : a_debabrata@yahoo.in



REF. NO : K-86 (OS)

DATE : 05-11-2016

NAME : Ms. Krishna Banerjee (OS)

SEX : F AGE : 70 YRS

Referred By : Dr. Ashim Chatterjee

H A E M A T O L O G Y

HAEMOGLOBIN : 13.6 gm/dL (93.8%) PCV : BLEEDING TIME (Ivy's method) :
(Cyanmethaemoglobin) [Male : 13.5 - 18 gm/dL]
[Female : 11.5 - 16.4 gm/dL]

WBC : 9,700 /c.m.m. MCV : [2-7 mins]

RBC : MCH : CLOTTING TIME (Lee and White):

PLATELET : 1.50 lakhs/c.m.m. MCHC : [4-9 mins]

RETICULOCYTE COUNT : RDW :

DIFFERENTIAL COUNT: Abs. Count Abs. Count

Neutrophil	: 65 %	6305 /c.m.m.	Blasts	: 0 %
Lymphocyte	: 32 %	3104 /c.m.m.	Promyelocyte	: 0 %
Monocyte	: 1 %	97 /c.m.m.	Myelocyte	: 0 %
Eosinophil	: 2 %	194 /c.m.m.	Metamyelocyte	: 0 %
Basophil	: 0 %		Band Form	: 0 %

E.S.R.

1st Hour :

FILM MORPHOLOGY

RBC : Normocytic, Normochromic.

WBC :

Platelets :

Parasites :

Checked By IPATH

Dr. Gupta
DCP MD (Cal)
Consultant Pathologist

Dr. A. Mukherjee
MBBS, MD (Path)
Consultant Pathologist

Dr. Adelene Basu
DCP, MD (Path)
Consultant Pathologist

Dr. S. Gupta
DCP MD (Cal)
Consultant Pathologist



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REF. NO : K-86 (OS)

DATE : 05-11-2016

NAME : Ms. Krishna Banerjee (OS)

SEX : F AGE : 70 YRS

Referred By : Dr. Ashim Chatterjee

B I O C H E M I S T R Y

TEST	TEST VALUE	REF. RANGE
SODIUM (NA) (ISE)	138.00 mEq/L	(135.00 - 155.00 mEq/L)
POTASSIUM (K) (ISE)	↓↓ <u>3.20 mEq/L</u>	(3.50 - 5.50 mEq/L)
<< LIVER FUNCTION TEST (LFT) >>		
SERUM BILIRUBIN (CONJ+UNCONJ) (Mod.Jendrassik-Grof)	0.86 mg/dl	(0.20 - 1.00 mg/dl)
Conjugated :	0.29 mg/dl	
Unconjugated :	0.57 mg/dl	
AST (SGOT) (Mod.UV-Kinetic method at 37°C)	56.00 IU/L	(Upto 40.00 IU/L)
ALT (SGPT) (Mod.UV-Kinetic method at 37°C)	53.00 IU/L	(Upto 40.00 IU/L)
ALKALINE PHOSPHATASE (IFCC Kinetic Method at 37°C)	79.00 U/L	(Upto 115.00 U/L)
SERUM PROTEIN (TOTAL) (BIURET)	7.30 gm/dl	(6.20 - 8.40 gm/dl)
Albumin :	3.80 gm/dl	
Globulin :	3.50 gm/dl	
Alb : Glb ::	1.1 : 1	

1

Contd ...

Dr. A. Mukherjee
MBBS, MD (Path)
Consultant Pathologist

Dr. Adelene Basu
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Immunopath diagnostic centre

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REF. NO : K-86 (OS)

DATE : 05-11-2016

NAME : Ms. Krishna Banerjee (OS)

SEX : F AGE : 70 YRS

Referred By : Dr. Ashim Chatterjee

B I O C H E M I S T R Y

TEST	TEST VALUE	REF. RANGE
SERUM UREA (KINETIC GLDH)	27.00 mg/dl	(10.00 - 45.00 mg/dl)
SERUM CREATININE (Jaffe's Alk. Picrate)	0.89 mg/dl	(0.70 - 1.30 mg/dl)
PLASMA GLUCOSE (FASTING) (GOD-POD)	81.00 mg/dl	(70.00 - 110.00 mg/dl)

N.B. : All reference ranges are age and sex matched. Reference limits mentioned herein are in accordance with the literature provided alongwith the kit which may change with the change in chemistry or the kit.

Checked By IPATH

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- 2) Mr. Roy (58 years old) was admitted with huge ascites and encephalopathy. He was diagnosed to have hepatocellular carcinoma with retroperitoneal and periportal lymphadenopathy. He was treated with Psorinum and other supportive medical management. Now the patient is doing his daily activities.
- 3) Mr. Lal Mohan Adhikary (86 years old) was admitted with left upper lobe mass with chest wall infiltration and rib erosion with mediastinal and hilar lymph nodes (CT proved). Histomorphological features are in favour of Non Small Cell Lung Carcinoma. He was treated with Psorinum Therapy in the clinic and after three months his pain and hemoptysis was reduced. PET CT proved that the mass lesion was arrested in the same dimension and no further extension found. He is now on Psorinum Therapy and other conservative management.

Many other patients with Lung CA, Renal Cell Carcinoma, Bone Metastasis etc. are treated successfully in the clinic.

Psorinum Therapy in future

The main issues of success of Psorinum Therapy are a) it is non-toxic b) easy to administer c) cheap d) minimum side effects. This issue will take this therapy in leading position in future. It can be given without specialist's (Oncologist) supervision. The main requirement is a team work of doctor of internal medicine, surgery and oncology for supportive treatment. There is a huge scope in the field of Bio-chemistry, Pharmacology etc. for research with Psorinum. The holistic approach to terminally ill cancer patients for their physical, emotional, practical problems should be associated with it. In our country like India where number of cancer patients are undiagnosed and refuse proper follow up due to financial cause and unawareness, Psorinum Therapy will take a leading role in future surely.

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Smiling both

AFTER CRITICAL OVERCOME (without RT or CT)

Dr. Sudipto Chatterjee & Mrs. Krishna Banerjee,

Dated : 9th November, 2016



INTERACTIONS

Dr. Asim Chatterje's Reply to Questions that are frequently asked

– By Mr. Sriman Chakraborty

Psorinum therapy is the basis of treatment here. How has this therapy been accepted all over the world?

There are different side-effects of cancer. As such the human body loses resistance power, infection spreads, excretion is disturbed, indigestion increases, small particles of blood decreases, respiratory problems aggravate. In that case, saline, antibiotics, rice-tube, minor surgeries are applied commonly as palliative management in order to remove the ailments inside the patients' body. Psorinum is one type of medicine which is consumed orally. It does not have any side-effects.

We have never claimed to possess such a medicine which will cure cancer. On the contrary we have specified the limitations of the conventional scientific treatment including the fact that it's too expensive. We have always tried to make the patient family aware of the future of this disease and help them to be practical enough not to spend all their possessions for a person who will not survive much longer on account of this deadly disease. Sometimes we explain them in a lighter vein with a smile, at times very crudely we depict the harsh reality depending upon the circumstances. The treatment procedure of cancer is so very complicated that despite of financial and technical support at the government hospitals they cannot handle properly and face many difficulties to treat cancer. Actually intensive care, wide experience and utmost devotion to the patient's cause is not always available. On the contrary Psorinum Therapy can be done with bearable cost. Usually when cancer was detected the patient and his relatives become wholly perplexed. They ran from pillar to post, one doctor to another, one institution to another institution. During this process they become fully exhausted – financially, mentally and physically. As a last resort they approach this centre with a frail health and empty pocket. Even in this terminal stage we apply our drug Psorinum along with the palliative treatment. Some amazing results were found when the patient gradually recovered and their life prolonged for years. This news percolates through the survived patients and their relatives to other victims. Its cost-effectiveness and painless experience attract the sufferers in general. The prolongation of the survival period and painless death through this therapy are its two unique characteristics by which it has been widely accepted all over the world. Patients from abroad also visit this centre and get good results.

How is it that you have managed to develop a cancer unit as a part of your residence? Have you depended on any government favour?

Yes, government cooperation has helped me to make this institution a recognized one. Government has permitted to seek the cooperation of many doctors and scientists. I have got

direct help from Chittaranjan National Cancer Institute. Specialized cancer physicians have also provided help. This is a problem too complex to be handled by any individual's initiative 120 crores of people that means the entire population in India should become aware of it. However, we never approached govt. for financial help. I have always preferred to be self-dependent. This is a recognized established organisation. So it can spread its wings on the entire region.

As far as we know Subodh Mitra Cancer Hospital developed the first terminal centre for cancer treatment in India. Why did you leave that Hospital?

Actually at Subodh Mitra Institute my total concept and palliative management of cancer could not be applied smoothly due to various reasons. When I left there the group of oncologists who were mainly concerned with allopathic mode of treatment were scared with the remarkable progress in curing the disease with Psorinum therapy. Lesser cost involved in this method also created discomfort to them. At a time they were apprehending that if this trend continues further then the oncologists at that centre could be unpopular very soon. So they tried to raise some plea or another to create their importance and thereby causing hindrance in smooth running of the institution. I could foresee the writings in the wall and to avoid further confrontation with this group. I decided to leave it peacefully. I preferred to work independently elsewhere where I can work without jealousy and the spirit of vengeance from anybody. However I shall always be grateful to the Board of Directors and my colleagues as the institute always cooperated with me. No matter what ephemeral misunderstanding and ego clashes might be there. I shall also always be thankful to my friends who had been with me in my critical time. I shall always be grateful to those who took me to Subodh Mitra institution.

We have noticed that in some cases, several institutions refuse for a surgery. But after a few months later surgery is done in your institution. How is it possible?

Yes, such cases are there. When conditions of tumour is so big that it does not permit a surgery, I apply Psorinum therapy, to reduce the size of the tumour. In many cases shrinkage in tumour take place and the patients are intended to sent to their respective institutions for necessary surgery. In most of the cases we find that people preferred to undergo surgery in this institution because of the low cost.

What causes or may cause cancer?

Most important factors are unhealthy and polluted atmosphere, genetic factor and different ways of tobacco consumption. Other factors include unnatural change in life-style, consumption of unhealthy food and artificial food products and extensive use of chemical colours in processed food. People who work with coal, plastic, asbestos, cement, chemical, sponge iron or pitch and when their skin get in direct contact with ultra-violet sun rays, they become prone to disease. At times usage of medicines based on chemical formulation for long time, may be the cause of cancer. Use of different male and female hormones without medical advice may cause cancer. Use of asbestos may cause pleural cancer, known as mesothelioma. Those engaged in making of asbestos and those who smoke a lot are prone to lung cancer. Those who work with synthetic colours may suffer from bladder cancer. People engaged in

operating x-ray and CT scan machines without any protection have serious chances to be affected by cancer, especially leukaemia. Cancer may also be caused from an ulcer in any part of the body if it faces friction repeatedly. Some viruses also cause cancer like leukaemia, Hodgkin's disease and malignant disease of lymphatic tissue in them. Some leukaemia is caused by hepatitis B and Hepatitis C. Imbalances in hormones do play an important role in escalating cancer.

What do you think about rendering help to the socio-economically backward section of the population? How do you apply the Psorinum Therapy?

500 year old research and success cannot be and should not be denied. Full treatment of cancer is yet not possible. We are trying the Psorinum therapy on CT, RT, and surgery resistant patients with Karnofsky scale below 50.

To render effective help to the socio-economically backward people we have concentrated on the non conventional ways of treatment of cancer to reduce the cost and making serious efforts to find ways can be viewed scientifically. Practically a huge infrastructure is needed to build up a full fledged cancer hospital. The first thing needed is a cancer detection centre followed by a group of expert doctors, huge manpower, general physicians, surgeons, radiologists, pathologists and surgical equipment like operation theatre, x-ray, USG, C.T Scan, MRI, Bone Scan etc. Oncologists and Oncosurgeons are needed after the disease is detected in order to decide whether the patient would have a major surgery or palliative surgery followed by Radiotherapy or Chemotherapy depending on the type of cancer. All these require plenty of money and time which we do not have. As a stop gap arrangement before the infrastructure can be built up we are on the look out to get the advantage for the poor regarding the existing arrangements especially in surgical area.

We analyze new patient whether he can accept the surgery. Our attempt aims at making the patient able to take the advantage of any existing surgical set up. Financially weak patients cannot afford surgery from private institutions. We conduct a counselling for them to make them agree to undergo a surgery through government hospitals. Similarly in cases like cervix, vocal cord cancer, RT produces positive result. Cases like – Lymphoma, Ovary cancer responses positively to CT. We thus arrange for providing government hospital membership cards to economically backward patients. Nevertheless we feel all these are very trivial attempts for a big and complex problem.

If thyroid cancer is detected in first stage, surgery, Chemotherapy, Radiotherapy, may be possible, but the palliative treatment is the only way out for those patients whose Karnofsky scale reaches below 50. It is sometimes very peculiar coincidence to observe that the ailing people who are financially weak are apparently found to be below Karnofsky scale. In that case cost-effective Psorinum therapy is the only way of treatment for these patients. It should run concurrently with palliative treatment. So depending on the financial condition of the patient we have to be careful to chalk out the plan and decide the mode of treatment.

Is cancer counselling absolutely necessary?

Cancer counselling is a very important issue. Generally curable disease like T.B. do not need counseling but cancer needs that because it is full of complexities. Day by day scientific

awareness of man is increasing Economic condition is also improving. These are accompanied by a general awareness of cancer. Since last 30 to 40 years, the world has become a family. Due to technical advancement people are getting informations of both development and crisis of the world. But to know the details some analytical reports and explanation may be necessary. Due to technical advancement people are getting information of both development and the crisis of the world. But to know the details some analytical reports and explanations may be necessary. Due to advancement of information technology, we come to know about the correlation between events.

In this context, cancer counselling means providing the patient with scientific information like what exactly has happened to him, what measures should be taken for that, how much would he be given only by a cancer specialist physician, because the entire issue is technical. But these points are not even easily understood by well-educated section of population of developing countries. 70% of the population do not possess higher education, 60% of the people are mostly from villages. Another question arises that how is it possible to cope with the complexities of cancer in the absence of properly qualified doctors in the villages.

Suppose a person is suffering from Hepato-cellular Carcinoma. He may be an ordinary man and may not be sufficiently educated. But he has come to cities for his treatment. It is very difficult to explain him about the disease. It is almost impossible for an Oncologist to explain what exactly is the patient suffering from and what is his treatment or what for has he come to the city or for how long would he have to stay in the city and also where would he stay. What treatment would he undergo and what would be the result. All queries which remain unanswered are to be answered satisfactorily.

Patient's initial counseling would be about the nature of the disease, whether the disease is of any specific type and how much the treatment would heal him/her. Another question arises on the patient's economic condition and the extent to which any financial aid would assist him. But there still remains a doubt about how much the patient would be benefited even after the conversation with the doctor. So the entire family should be brought into confidence. Counseling in Western countries are entirely different from that of India. In our country close relatives get much involved with the treatment procedure. Unlike western countries efforts must be made to develop more interactions with the relative who extend their hand of cooperation in this distress of the patient. Different categories of middle class people try to save the life of their loved one at the cost of their own properties. But still patients may not survive. So this financially backward section of the population do not consider cancer as an individual problem but a menace to the entire social development. They tend to fight against it by mutual understanding and co-operation with each other and the Doctor with all his experience and knowledge should stand by them during the process of counseling. Medical science at present has become very commercial. In that case, when we come to a complex and highly technical expensive treatment, the patient's family urgently needs healthy advice and proper counselling. Their spontaneous discussion with doctors,

nurse and pathologists are essential and most welcome.

How far we are prepared to face the Epidemic of Cancer?

At present, the most severe problem of the world is the widespread deadly disease of cancer. World Cancer Report do claim that by 2020 the number of affected cancer patients would increase to almost 15 million. So public health issues should be mostly concerned with the treatment of this disease as till date no such drug has been discovered which would cure it completely. The rate of increase of cancerous patients indicates that the spread of the disease is taking the shape of an epidemic. One statistics show that out of 56 million deaths in 2000 12% was due to malignant tumour. Many countries witness 14% of their deaths due to cancer. In 2000 5.3 million men and 4.7 million women had cancer and 6.2 million died. Thus the World Cancer Report considers cancer to be a serious problem to the future of human health development. Both the developed and the developing countries are mostly affected by the disease. Asian countries like India, China, Bangladesh, Pakistan are expected to be the greatest victims. If medical science fails to treat and cure this disease then the entire human civilization would be jeopardized. The most important limitation is still the absence of an adequate drug for the Disease. There are 120 different varieties of cancer. In certain circumstances, Chemotherapy, Radiotherapy or surgeries help to reduce pain and increase life span of the cancer patients.

Although some treatment is possible in the initial or second stage of the disease but medical science fails to treat the third or fourth stage. Nevertheless some cancer patients may at times do survive for five or seven years without any major treatment. Though, some types of cancer causes death within a few months even after major attempts to treatment.

Incidentally all technical processes are invented by the Western countries, as such they are very expensive and much beyond the reach of common people of our country. This situation demands immediate implementation for some pre-emptive measure to avoid the imminent devastating onslaught of the disease on the common man our land.

Primarily patients are to be selectively ramified considering his financial standing, health performance status, physical resistance to toxic drug and other feature as well. If it found neither his finance permit nor his health condition allow to continue conventional treatment emphasis should be given to palliative management from the very inception.

As a strategy against this epidemic, apart from taking other necessary measures, emphasis should be given to some particular varieties of this disease like Oral cancer which is the most widespread type in India. It takes much severe form which makes it difficult for the patient to move from one place to another and even causes sudden death of the patient. As a preventive measure anti-tobacco movement should be launched together with mobilizing our human resources like unemployed dental-surgeons who are eagerly waiting their gainful employment. Statistics show that there are about 25000 yearly pass outs of dental surgeons from both government and private dental hospitals but they mostly remain unemployed. If they can be properly trained about oral cancer then the disease can be largely prevented and can be taken care of through their gainful employment as well. This awareness together with other adequate training would also help in early detection of Breast Cancer, Cervix cancer

and cancer of vocal chord. In this way other ronts can be opened to blunt the attack of the epidemic in our country and cancer patients can be treated better and their life span can be expanded.

In this repertory, we have proposed the model of Mini Cancer Unit to be attached with every health centre throughout the length and breadth of our country. This model spells out the concrete steps of fighting the impending epidemic at a reasonable cost.

Why you gave emphasis to have a panoramic view of the entire problem of cancer?

A doctor is concerned about the patient, medicine and treatment and a scientist on the other hand is thoughtful about the disease and the scientific application of new medicines on different diseases. But in-depth study always felt the need of a comprehensive idea and a panoramic view of the entire problem to find out the realistic and workable solution commensurate with the need of the situation.

You have mentioned earlier about certain measures to be taken under palliative treatment like feeding jejunostomy, colostomy, stenting etc. Will you kindly throw some light on it?

To be very frank it is difficult to explain these terminologies in one or two words. However, for realization of a layman it can be narrated as such.

When we consume food and water through our mouth it reaches the stomach from where it under goes metabolism and reaches the colon. Thereafter from colon the waste remains reaches the rectum and is called the stool or faeces, which is then excreted from the body. This process is hampered or obstructed if there is a tumour in the food pipe. As a result food cannot pass. The patient is then made to consume food through rice-tube. This is a palliative treatment, though this is related with the treatment of cancer. But the patient can be provided with food for some days through the rice-tube.

After a few days this method usually becomes inapplicable. Now the process of feeding by way of jejunostomy arises. A hole is made below the stomach to fix a tube there through which a patient can be provided with food for some days. At times there may be a permanent surgery, when the entire tumour in the Oesophegus is removed. This process is effective only if the tumour is detected at the primary stage. This is still a major operation. When the tumour is enlarged it cannot be removed. Temporary arrangements are made in the adjoining regions of the stomach to enable the patient to eat which is also regarded as bypass surgery or stenting. When tumour is detected at a primary stage in colon, then also a major portion of the colon along with the tumour can be removed. This is called hemi-colostomy. This is surgery and not palliative treatment. Again when the tumour is abnormally enlarged it is not removed by surgery, instead in the adjoining areas a pathway is made to bypass the obstructions in the way inside the body. This is called Colostomy and this comes under the jurisdiction of palliative treatment.

CRITICAL OVERCOME



CRITICAL OVERCOME EPISODE 1

The years 1995 and 1999, both are memorable to me. The reason being the work that we had been doing since the 1980s received its due acknowledgement from the scientific community which eventually got documented in the West Bengal Chapter by 1995, after a series of phases i.e. decision period, study period and war period. In December 1993, my research got documented in the Indian Science Congress under the banner of the prestigious School of Tropical Medicine. The paper that I presented in the Rajasthan Session of the Indian Science Congress consisted of many reputed names from the Oncology fraternity of West Bengal like Prof. Subir Dutta, former-Dean of Medical Faculty, Calcutta University and a famous pathologist, Prof. Amiyo Kr. Hati, former-Director of the School of Tropical Medicine, Prof. R. N. Brahmachari and many other famous oncologists, onco-surgeons and onco-scientists of that time. With this the war period, I have repeatedly referred to earlier, had come to rest by the end of the year 1994. In 1995 itself one of my papers was published in the Medical-bulletin of the School of Tropical Medicine. This clearly means that my efforts had also received institutional acceptance by that time.

Many of our other publications that were presented at various scientific-forums under the banner of School of Tropical Medicine, most of them were documented in the twelve years work of School of Tropical Medicine. At the same time I was invited to read a paper at the 2nd West Bengal State Science Congress in the seminar hall at the Indian Institute of Chemical Biology, Kolkata. This seminar was organized by the joint initiative of the West Bengal Directorate of Science and Technology, West Bengal Government and the Calcutta University. The chairperson of the committee which judged this seminar was Prof. Amar Bhaduri and an eminent member of the panel was Prof. Gauripada Dutta. There I had presented a tabular representation of 54 cancer patients under my treatment and also showed the total regression of cancer in two patients – one advanced stomach cancer patient who was turned down by the Chittaranjan National Cancer Institute, Mr. Bijoy Kr. Hui, and another advance Gall Bladder Cancer and liver metastasis patient, Mrs. Binapani Sarkar. There I had to face many questions but since the medium of communication was Bengali I could comfortably answer all their questions. At the end of my presentation, the people attending that session appreciated my work and efforts whole-heartedly. Later the entire paper was published in the book titled '2nd West Bengal State Science Congress' under my authorship by the Calcutta University. On that platform I represented my small NGO 'The New Resource' and till date it is a matter of pride for me.

Cancer research in doldrums

Prestige meet minus allopathic representative from city

BAPPAMAJUMDAR

Conventional cancer research here has all but hit a dead-end. Case in point: not a single allopathic doctor from the city or the state has been invited to the international conference on cancer research, being held in Delhi from Sunday.

Instead, it is a homoeopath, Asim Chatterjee of Lake Town, who will represent Bengal at Watch 2004, organised by World Health Organisation (WHO), the European Society of Medical Oncology and others like the International Congress on Oral Cancer and the International Association for the Study of Lung Cancer.

"This is a unique cancer-research conference, where most of

the participants are from abroad. And only the best work will be presented," says A.K. Varma, convenor of the conference and former surgeon to the President of India.

Cancer experts in Bengal admit that "lack of motivation, opportunity, and facilities" hinder cancer research.

"Our young researchers do not have an opportunity to go for exchange programmes abroad to learn how research work is conducted there," complains Anup Majumdar, head of radiotherapy at SSKM Hospital.

There are many "top" cancer

ence," says Gangopadhyay. "I think what has happened (*vis-à-vis* Watch 2004) is a stark reminder that it is time to do something about it."

Asish Mukherjee, director of Subodh Mitra Memorial Cancer Hospital, blames the "lack of infrastructure" for slowing down cancer research in the state.

In Calcutta and the rest of Bengal, around 60,000 new cases of cancer are reported every year. Treatment is an option only at a few centres and no research of note is being carried out.

"Not much work has been done here. But we are now creating the right atmosphere to encourage more cancer research," promises C.R. Maiti, director of medical education.

specialists in the city, some of whom have spent years abroad or in leading hospitals of Mumbai and New Delhi. But no one has made it to the Watch 2004 list, that includes researchers in oral, laryngeal and oesophageal cancer.

Subir Gangopadhyay, head of the radiotherapy department in Medical College and Hospital, admits that patient pressure and the long hours of practice prevent them from pursuing any research work.

"It is true that we don't have enough quality work to be presented at an international confer-

দ্বিতীয় পশ্চিমবঙ্গ রাজ্য বিজ্ঞান কংগ্রেস

কলকাতা, ২৮শে ফেব্রুয়ারি—২রা মার্চ, ১৯৯৫

গবেষণাপত্র

পশ্চিমবঙ্গ রাজ্য বিজ্ঞান ও প্রযুক্তি সংসদ
বিজ্ঞান, প্রযুক্তি এবং অপ্রচলিত শক্তির উৎস বিভাগ
পশ্চিমবঙ্গ সরকার
এবং
কলিকাতা বিশ্ববিদ্যালয়

সূচিপত্র

বিষয়	পৃষ্ঠা
1) উদ্ভিদ বিজ্ঞান (Botany)	১—১৩
2) কৃষি বিজ্ঞান (Agriculture)	১৫—৭৫
3) খাদ্য প্রযুক্তি (Food Technology)	৭৭—১০৭
4) গণিত (Mathematics)	১০৯—১৪৪
5) চিকিৎসা বিজ্ঞান (Medical Science)	১৪৫—১৯০
6) প্রাণ রসায়ন (Biochemistry)	১৯১—২৯৭
7) পরিবেশ ও স্বাস্থ্য (Environment & Health)	১৯৯—২১৪
8) প্রাণিবিদ্যা (Zoology)	২১৫—২৪৭
9) ভূতত্ত্ব (Geology)	২৪৯—২৭০
10) পদার্থবিদ্যা (Physics)	২৭১—২৯৯
11) রসায়ন ও প্রযুক্তি (Chemistry and Technology)	৩০১—৩৪৫
12) সমাজ ও বিজ্ঞান (Society and Science)	৩৪৭—৩৯৩

কার্সিনোমা চিকিৎসায় একটি প্রচলিত হোমিওপ্যাথি ওষুধ 'সোরিনাম'-এর অপ্রচলিত পদ্ধতিতে পরীক্ষামূলক ব্যবহার—দুজন রোগীর উপর প্রয়োগের ফলাফল

অসীমকুমার চট্টোপাধ্যায়

বি নিউ রিসোর্স, কলকাতা

৫৪ জন বিভিন্ন ধরনের ক্যান্সার রোগীর চিকিৎসায় একটি প্রচলিত হোমিওপ্যাথি ওষুধ 'সোরিনাম'-এর অপ্রচলিত পদ্ধতিতে পরীক্ষামূলক ব্যবহার করা হয় এবং সর্বাধুনিক নির্ণয় পদ্ধতির দ্বারা পর্যায়ক্রমে এই পরীক্ষার ফলাফলগুলির বিজ্ঞানসম্মত মূল্যায়ন করা হয়। এই পরীক্ষা করেকজন বিশিষ্ট আলোপ্যাথি চিকিৎসক, তৈব রসায়নবিদ এবং কয়েকটি প্রতিষ্ঠিত গবেষণামূলক প্রতিষ্ঠানের সাহায্য নেওয়া হয়। ক্যান্সার বিশেষজ্ঞদের দ্বারা প্রত্যাখ্যাত কয়েকজন ক্যান্সার রোগীর উপর এই পরীক্ষামূলক চিকিৎসাপদ্ধতি প্রয়োগের ফলে আশ্চর্যজনক উন্নতি লক্ষ করা যায়। এই গবেষণাপত্রে পরীক্ষিত দুইজন রোগীর চিকিৎসার বিবরণ সম্পূর্ণ তথ্যসহ পরিবেশিত হল।

ক্যান্সারের চিকিৎসায়, বিশেষত এডিনো কার্সিনোমার ক্ষেত্রে 'সোরিনাম' অপ্রচলিত পদ্ধতিতে ব্যবহার করে কতটা কার্যকরী হতে পারে, তা নির্ণয়ের জন্য বিভিন্ন ক্ষেত্রের বিশেষজ্ঞ চিকিৎসক ও গবেষকদের ব্যাপক অনুসন্ধান বিশেষভাবে প্রয়োজন।

বর্তমান পৃথিবীতে ক্যান্সার রোগ সমগ্র মানবজাতির কাছে একটা ভয়াবহ অভিশাপ আর চিকিৎসা বিজ্ঞানীদের কাছে এ রোগের নিরাময় হল একটা বিরটি চ্যালেঞ্জ কারণ এ বিষয়ে এখনও সর্বসম্মত কোনও পথনির্দেশ পাওয়া যায়নি।

ক্যান্সারের চিকিৎসায় সার্জারির একটি বিশেষ ভূমিকা আছে। প্রধানত দুটি কারণে সার্জারির প্রয়োজন হয়—(১) রোগ নির্ণয়ের জন্য এবং ক্যান্সার আক্রান্ত শরীরে বিশেষ অংশের নমুনা সংগ্রহের জন্য, (২) আক্রান্ত অংশকে শরীর থেকে বাদ দেওয়ার জন্য।

রেডিওথেরাপি ও কেমোথেরাপি ক্যান্সার চিকিৎসায় সাহায্য করে। তাই সার্জারি, রেডিওথেরাপি ও কেমোথেরাপি কখনও পৃথকভাবে আবার কখনও বা একই সঙ্গে ক্যান্সার রোগ নির্ণয় ও চিকিৎসায় ব্যবহার করা হয়।

শরীরের বিশেষ কিছু অংশের বিশেষ ধরনের ক্যান্সারের চিকিৎসার জন্য যেমন খাদ্যানালী, শ্বাসনালী, জিহ্বা, অস্থি সংক্রমণ, যন্ত্রণা উগ্গশম, 'প্যাথোলজিক্যাল হ্রাসকারণ' প্রতিরোধ, রেডিওথেরাপির ব্যবহার প্রয়োজন হয়। অবশ্য ফুসফুস বা শরীরের অন্যান্য অংশে ক্যান্সার প্রতিরোধ রেডিওথেরাপি ছাড়াও অন্য ব্যবস্থা নেওয়া যায়।

লিউকেমিয়া, লিম্ফোমা ও তৃতীয় ক্যান্সার চিকিৎসায় প্রাথমিক পর্যায়ে কেমোথেরাপি প্রয়োগ করা হয় জি আই ট্রাক ও স্তনের ক্যান্সারের চিকিৎসায় সার্জারি ও কেমোথেরাপি একই সঙ্গে ব্যবহার করা যায়।

কেমোথেরাপি ব্যবহারের ক্ষেত্রে শরীরের কিছু প্রাথমিক ও মৌলিক মান নির্ধারণের প্রয়োজন হয়। ক্যান্সার রোগীর শরীরের বিভিন্ন অংশ ও সংলগ্ন কোষগুলির কার্যকারিতা বজায় রাখার জন্য ওষুধ ব্যবহার করা হয়। তবে এ সময়ে এক বা একাধিক সম্ভাব্য প্রতিক্রিয়ার বিচ্ছেদও জরুরি হয়ে পড়ে। বিশেষ বিশেষ ক্ষেত্রে রোগীর অবস্থা এই 'হোস্টফ্যাক্টর'

(প্যারামিটার)গুলির সঙ্গে বিশেষ গুরুত্বপূর্ণ হয়ে ওঠে। আসলে কেমোথেরাপি প্রয়োগের বিষয়টি অনেকাংশেই নির্ভর করে ক্যানসার আক্রান্ত রোগীর শারীরিক কার্যকরী ক্ষমতা, ('পারফরম্যান্স স্টেটাস'), প্রতিরোধ ক্ষমতা, পুষ্টি, দেহের জৈব রাসায়নিক প্রক্রিয়া, রক্তের উপাদান ও তার স্বচ্ছতা এবং নিভারণের কার্যকারিতা নির্ধারক রক্ত পরীক্ষার ফলাফলের উপর।

করনোফেসকি স্কেল অনুযায়ী ক্যানসার রোগীর শরীরের 'পারফরম্যান্স স্টেটাস' ৫০ (পঞ্চাশ) এর নীচে থাকলে কেমোথেরাপি প্রয়োগ করা যায় না। অন্যভাবে বলা যায়, এই স্কেল অনুযায়ী ৫০ (পঞ্চাশ) এর উপরেই কেমোথেরাপির প্রয়োগ সীমাবদ্ধ। ওই অবস্থায় রোগী অসুস্থ হলে বা যে কোনভাবে সক্ষমতা হারালে রেডিওথেরাপি বা কেমোথেরাপি কোনমতেই প্রয়োগ করা যায় না। শুধুমাত্র রোগীর সাময়িক স্বাচ্ছন্দ্যের প্রয়োজনে যন্ত্রণা উপশমের ওষুধ, হিমোগ্লোবিনের প্রয়োজনে রক্ত, শরীরের জলীয় অংশের সমতা রক্ষার জন্য গ্লুকোজ, লবণ জল প্রভৃতি ব্যবহার করা যায়।

ক্যানসার চিকিৎসায় হোমিওপ্যাথি ওষুধের কার্যকারিতা বিশ্লেষণ প্রসঙ্গে একটি গবেষণাপত্র সিদ্ধান্ত উল্লেখ করা প্রয়োজন। করনোফেসকি স্কেল অনুযায়ী শারীরিক 'পারফরম্যান্স স্টেটাস' ৫০ (পঞ্চাশ) এর নীচে এবং কেমোথেরাপি বা রেডিওথেরাপি করা সম্ভব হয়নি এমন ৫৪ জন ক্যানসার রোগীর প্রচলিত চিকিৎসা বজায় রেখেও হোমিওপ্যাথি ওষুধ 'সোরিনাম' অপ্রচলিত পদ্ধতিতে বিশেষ মাত্রায় (পোটেনসি) ও পরিমাণে ব্যবহার করে উল্লেখযোগ্য ফল পাওয়া সম্ভব।

উপাদান ও প্রয়োগপদ্ধতি :

বিভিন্ন ধরনের ক্যানসার রোগীজ্ঞাত যে ৫৫ জনের উপর হোমিওপ্যাথি ওষুধ 'সোরিনাম' ব্যবহার করা হয় তাদের চিকিৎসা অনেক আগেই শুরু হয়েছিল প্রচলিত চিকিৎসাপদ্ধতি অনুসরণ করে। এমনকি অত্যন্ত নির্ভরযোগ্য একাধিক প্রতিষ্ঠানে অভিজ্ঞ ও বিশেষজ্ঞ চিকিৎসকদের তত্ত্বাবধানে সর্বাধুনিক প্রযুক্তির সাহায্যে (যেমন রেডিও ইমেজিং, আলট্রাসোনোগ্রাফি, সি টি স্ক্যান, এন্ডোস্কোপি, সাইটোলজি, হেমাটোপ্যাথলজি) একাধিক পরীক্ষার মাধ্যমে তাদের ক্যানসার রোগ চূড়ান্তভাবে নির্ণয় করা হয়েছিল এরপর একই চিকিৎসাপদ্ধতি অনুসরণ করে রোগীদের উপর পর্যায়ক্রমে অ্যান্টিবায়োটিক, অ্যামিনোড্রিপি, ফাট সলিউবল, রক্ত ও স্যালাইন ব্যবহার করা হয়। এর অনেক পরে 'সোরিনাম' ওষুধটি বিশেষ ও অপ্রচলিত পদ্ধতিতে ব্যবহার করা হয় এবং ওষুধটির কার্যকারিতা বজায় রাখার জন্য প্রচলিত পদ্ধতির প্রয়োগও সমান্তরালভাবে বজায় রাখা হয়। এমনকি কয়েকজন ডায়বেটিক ক্যানসার রোগীর ক্ষেত্রে ইনসুলিনের প্রয়োগও অব্যাহত থাকে। কিন্তু কোনও অবস্থাতেই রোগীদের কারও উপর রেডিওথেরাপি বা কেমোথেরাপি করা হয়নি।

চিকিৎসার বিভিন্ন পর্যায়ে ক্যানসার রোগীদের অবস্থা-সংক্রান্ত প্রয়োজনীয় তথ্যগুলির আনুপূর্বিক সংগ্রহ ও সংরক্ষণে সর্বাধুনিক প্রযুক্তির সাহায্য নেওয়া হয়। বিভিন্ন ধরনের ক্যানসার রোগীর ওষুধ নির্বাচন ও সেগুলির প্রয়োগ সম্পর্কে প্রতিটি ক্ষেত্রে সংশ্লিষ্ট বিষয়ের বিশেষজ্ঞের পরামর্শ নেওয়া হয়। এই পর্যায়গুলি রোগীর শারীরিক সক্ষমতা, রোগের উপশম ও ক্রমোন্নতির বিষয়ে প্রাসঙ্গিক তথ্য সংগ্রহ করা হয়। 'সোরিনাম'-এর পরিমাণগত ও মাত্রামানকে পর্যায়ক্রমে প্রয়োগের পরপরই প্রত্যেকটি রোগীর জৈব রাসায়নিক অবস্থা-সংক্রান্ত মান ক্রমান্বয়ে পর্যবেক্ষণ করা হয়।

87. LILIAN SARANI,
CALCUTTA-INDIA. Sec 2070
RESIDENCE 75-1255

F.N.A.L. - From his man
closed gate behind.
Pony clip. Car. horn - 3 Bays
- dt 22. 4. 90 -

Adm: - Blood spec for Hb, T. Co + Phlebotomy
L.F.T

Ph. hyal. *Ph. hyal.* i. *Ph. hyal.*
i. *Ph. hyal.* i. *Ph. hyal.*
Ph. hyal. i. *Ph. hyal.*

Leica in Bremen

Notes: - Chem. Comp. (14 or 20)

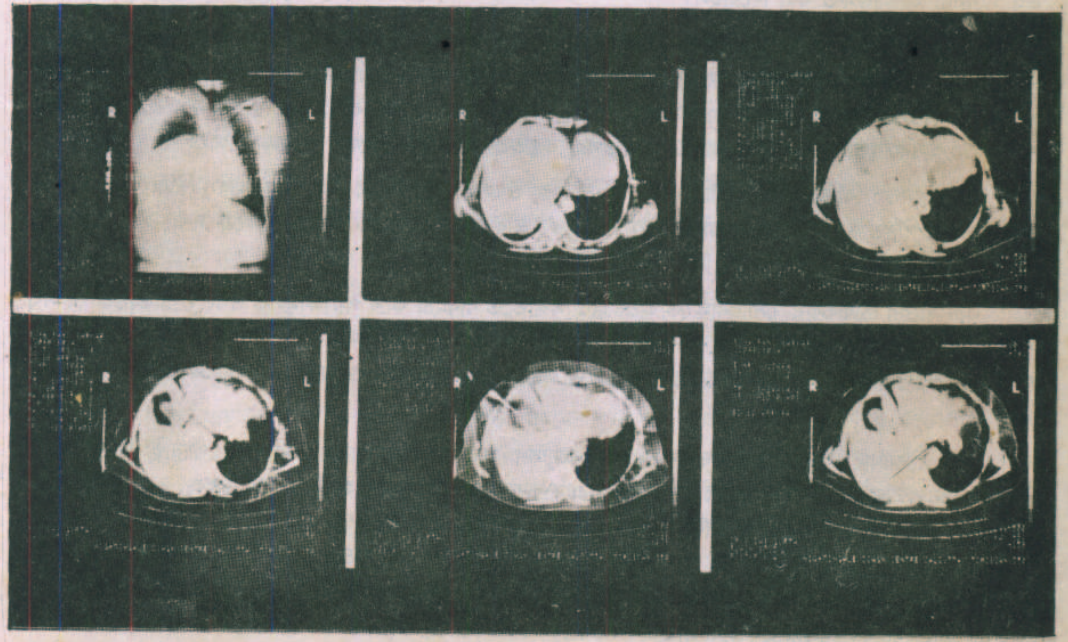
- Eye - Fair
- Hair -
- N.P.
- 70
- 100 - 1000

3 slide box has
lines for series at
2. Lane change box, Row

Ly. flavus (100g) - 10g
- Cal. lencostri (10g) - 10g ✓
drying @ 5%. sent to Delp
for Zelp.

261
100. 2. 5000





292

ফলাফল ও আলোচনা :

সাধারণত কর্নোফোসকি স্কেলে ৫০ (পঞ্চাশ) এর নীচে অবস্থানকারী ক্যানসাররোগীদের ক্ষেত্রে কেমোথেরাপি অথবা রেডিওথেরাপি প্রয়োগ করা যায় না।

আবার প্রাথমিক অবস্থাতেও, বিশেষ করেকটি ধরনের ক্যানসার ছাড়া, কেমোথেরাপি বা রেডিওথেরাপি বিশেষ কার্যকর হয় না।

‘আডভান্স স্টেজে’ ও ক্যানসার রোগীদের ক্ষেত্রে সার্জারি সম্ভব হয় না।

পরীক্ষালব্ধ ফলাফলের বিশ্লেষণে দেখা যায়, কর্নোফোসকি স্কেল পরিমাপ অনুসারে যে সব ক্যানসার রোগীদের অবস্থান ছিল ৫০ (পঞ্চাশ) এর নীচে সেই ৫৪ জন রোগীর প্রত্যেকের চিকিৎসায়, বিশেষ বিশেষ অবস্থায় ‘সোরিনাম’ বিশেষ মাত্রা ও পরিমাণে প্রয়োগ করে উল্লেখযোগ্য ফল পাওয়া সম্ভব (সারণি দ্রষ্টব্য) এই গবেষণাপত্রে সংযোজিত সারণির অন্তর্ভুক্ত দু’জন ক্যানসার রোগীর এই পদ্ধতিতে চিকিৎসার তথ্যভিত্তিক বিবরণ পূর্ণাঙ্গভাবে দেওয়া হল (সংযোজনপত্র দ্রষ্টব্য)।

এ যাবৎ অন্য প্রচলিত পদ্ধতিতে বা বাস্তবায়িত হয়নি হোমিওপ্যাথি ওষুধ প্রয়োগের ফলে ক্যানসার আক্রান্ত রোগীদের শারীরিক অবস্থার পর্যায়ক্রমিক উন্নতি ও পরমায়ু (লাইফস্প্যান) বৃদ্ধি সম্ভব হয়েছে।

এই গবেষণা প্রাথমিক পর্যায়ে ছিল অসংগঠিত। তাই প্রধানত মুমূর্ষু ক্যানসার রোগীদের উপর প্রচলিত হোমিওপ্যাথি ওষুধ ‘সোরিনাম’ এর অপ্রচলিত পদ্ধতিতে ব্যবহারের কার্যকারিতা সংক্রান্ত গবেষণার কাজ চালাতে হয়েছিল। বিভিন্ন ধরনের ক্যানসার রোগীদের উপর, যাদের প্রচলিত চিকিৎসার সর্বশেষ স্তর পর্যন্ত নিয়ে গিয়ে বাতিল ঘোষণা করা হয়েছিল, এই গবেষণা আকাঙ্ক্ষিত ফল দিয়েছিল।

গবেষণার দ্বিতীয় পর্যায়ে গবেষণাক্ষেত্রটি সীমিত করা হয়েছে। শুধুমাত্র স্টম্যাক, লিভার, গলব্লাডার, প্যানক্রিয়াস এবং সার্জারি সম্ভব নয় এমন ফুসফুসের ক্যানসারের নিরাময় প্রচেষ্টার মধ্যেই বর্তমান গবেষণার পরিধি চিহ্নিত। এ ছাড়া প্রযুক্ত ওষুধটির ফার্মাকোডাইনামিক্স ও ফার্মাকোকাইনেটিক্স গবেষণা চলছে। ওষুধটির জৈব রাসায়নিক বিক্রিয়া ও সেই সম্পর্কিত কার্যকারিতা জানার জন্য প্রাণীদেহের উপর বিজ্ঞততর পরীক্ষা-নিরীক্ষার প্রয়োজন আছে।

‘বিজ্ঞান-নির্ভর বিভিন্ন ক্ষেত্রে মৌলিক গবেষণায় যুক্ত উৎসাহী সুবীজনের এবং অনুসন্ধিৎসু চিকিৎসাবিজ্ঞানী ও বিশেষজ্ঞ সুসংগঠিত ও সৃষ্টিধর্মী কর্মপ্রয়াসের সংযুক্তিই হয়ত ভবিষ্যতে ক্যানসার নিরাময়ের ক্ষেত্রে দেখাতে পারে কোনও আলোকমাত্র পথনির্দেশ।

ক্যানসারের চিকিৎসায় ‘সোরিনাম’কে অপ্রচলিত পদ্ধতিতে ব্যবহার করে পরীক্ষামূলক এবং তথ্যভিত্তিক কোনও গবেষণা এ পর্যন্ত হয়নি। তাই এই গবেষণাপত্রের সঙ্গে কোনও প্রামাণ্য সূত্রনির্দেশ সংযোজন করা সম্ভব হল না। এই অর্থে গবেষণাপত্রটি মৌলিক বলে ধরে নেওয়া যেতে পারে। এজন্য এখানে শুধুমাত্র গবেষণালব্ধ তথ্যের সংযোজন করা হল।

এই গবেষণার কাজে প্রত্যক্ষভাবে যাদের অনুগ্রহ সাহায্য ও মূল্যবান পরামর্শ পেয়েছি তাঁরা হলেন ডঃ আর এন ব্রহ্মচারী, ডঃ সুবীর কুমার দত্ত, ডঃ আর এস ভক্ত, ডঃ অমিয়কুমার হাটি, ডঃ প্রবীর কুণ্ডু, ডঃ বর্ণা ভট্টাচার্য, ডঃ মলয় পরামাণিক, ডঃ আই চ্যাটার্জি, ডঃ হিরণ্ময় মুখার্জি, ডঃ মধু দত্ত চৌধুরী, ডঃ বিষ্ণুপ্রসাদ মুখার্জি, ডঃ প্রণব ভট্টাচার্য, ডঃ ডি সি দাস, ডঃ পার্থ বানার্জি, ডঃ সুব্রত ভট্টাচার্য, ডঃ সুভাষ ঘোষাল, ডঃ অশোকানন্দ কোনার, ডঃ সম্রাট ভট্টাচার্য, ডঃ সত্যপ্রিয় দে সরকার, ডঃ বিশপতি মুখোপাধ্যায় ও ডঃ উৎপলা চট্টোপাধ্যায়। বিভিন্নভাবে অকুণ্ণ সাহায্যে কলকাতার যে সব প্রতিষ্ঠান আমাকে ঋণী করেছে সেগুলি হল চিত্তরঞ্জন ক্যানসার হাসপাতাল,

এন আর এস মেডিক্যাল কলেজ ও হাসপাতাল, আদেসিসিয়েশন ফর দি কন্টিভেনশন অফ সায়েন্স, স্কুল অফ ট্রপিক্যাল মেডিসিন, ইউনিভার্সিটি কলেজ অফ মেডিসিন, মেডিক্যাল কলেজ হাসপাতাল, ন্যাশনাল মেডিক্যাল কলেজ ও হাসপাতাল এবং এস এ কে এম কলেজ ও হাসপাতাল। আর চিকিৎসা বা ক্যানসার গবেষণার থেকে যোজন দূরত্বে অবস্থান এমন অসংখ্য সাধারণ মানুষের অন্তর্হীন উৎসাহ, ভালবাসা আর প্রেরণা সবসময়ই আমার কাছে অর্থবহ হয়েছে তাদের আন্তরিক প্রত্যাশায়—সাফল্য আর বেশি দূরে নয়।

রক্তকঠিন ব্যয়বহুল উচ্চ প্রযুক্তিনির্ভর ক্যানসার রোগের চিকিৎসা ও গবেষণার দূয়ার আজ প্রায় অর্গলবদ্ধ। সারা পৃথিবী জুড়ে চেষ্টা চলেছে এই দুরারোগ্য ব্যাধির কারণ নির্ণয় ও দ্রুত সম্পূর্ণ আরোগ্যলাভের পথ অন্বেষণের। তৃতীয় বিশ্বের দেশগুলিতে (যেমন ভারত, চীন, মিশর) প্রচলিত আছে বিভিন্ন ধরনের প্রাচীন চিকিৎসা পদ্ধতি। চিকিৎসার কোনও বিশেষ একটি বা একাধিক পদ্ধতি ও প্রক্রণের সমন্বয়ে ওই সব পদ্ধতির যাচাই করে নেওয়া আজ বোধ হয় অপরিহার্য হয়ে উঠেছে। সর্বাধুনিক প্রচলিত পদ্ধতির তুলনায় বিচার-বিশ্লেষণের সীমানাকে অতিক্রম করে পারস্পরিক সহযোগিতায় বুদ্ধি, মনন ও নির্মোহ দৃষ্টিভঙ্গি প্রয়োগ এটি যাচাই করার কাজটি সহজ ও সার্থক করে তুলবে বলে আমার দৃঢ় বিশ্বাস।

সংযোজন পত্র : (১)

বীণাপাণি সরকার ৫১ বছর বয়সের এক ভদ্রমহিলা। ২৫.৩.৯৪ তারিখে পেটের ডানদিকের উপরের অংশে যন্ত্রণা ও ফোলা নিয়ে কলকাতার মেডিক্যাল কলেজ ও হাসপাতালে ভর্তি হন। ডাক্তারি পরীক্ষায় পেটের ডানদিকের উপরের অংশে একটি অর্বুদ ধরা পড়ে। ই সি জি স্কোলে এর স্কের ছিল (৪) চার। এ ছাড়া সি টি স্ক্যানে লিভার ও গলব্লাডারের পরস্পর সংযুক্ত মডুলার এনলার্জমেন্ট ও অ্যাসাইটিস দেখা যায়। ২২.৪.৯৪ তারিখে লিভার ও গলব্লাডারে সি টি গাইডেড বায়োপসিতে ধরা পড়ে এডিনোকার্সিনোমা। রোগিণীকে কেমোথেরাপি করার পরামর্শ দেওয়া হয়। কিন্তু তিনি তাতে রাজী হন না। ৫.৫.৯৪ তারিখে রোগিণীর উপর প্রচলিত হোমিওপ্যাথি ওষুধ 'সোরিনাম' অপ্রচলিত ও বিশেষ পদ্ধতিতে প্রয়োগ করা শুরু হয়। ধীরে ধীরে যন্ত্রণার উপশম হয়। অ্যাসাইটিস ও অর্বুদটি বিলীন হতে শুরু করে। ক্রমশই রোগিণীর স্বাস্থ্যের উন্নতি লক্ষ করা যায়। ই সি এ জি স্কোলে রোগিণীর স্কের হয় ০ (শূন্য)। এরপর ২.৯.৯৪ তারিখে নিউক্লিয়ার স্ক্যান পরীক্ষায় লিভার ও গলব্লাডারে কোনও অর্বুদ এবং অ্যাসাইটিসের চিহ্ন পাওয়া যায়নি। পরবর্তীকালে ২৫.১১.৯৪ তারিখে লিভার পরীক্ষা (ফাংশনাল টেস্ট) করে কোনও অসঙ্গতি ধরা পড়েনি।

সংযোজন পত্র : (২)

বিজয় কুমার হুই ৭৫ বছর বয়সের এক ভদ্রলোক। ১৯৮৫ সালে পাকস্থলীতে ঘা হওয়ার জন্য তাঁর পাকস্থলীর সঙ্গে ছোট অস্ত্র জুড়ে দেওয়া হয় (গ্যাসট্রোজুন্স্টমি)। সে সময় তাঁর পাকস্থলীতে ক্যানসার দেখা যায়নি। ১৯৯২ সালে ক্ষুধামন্দা, বমি, ওজনহ্রাস ও রক্তপায়খানার জন্য তাঁকে নার্সিংহোমে ভর্তি করা হয়। তখন গ্যাসট্রোস্কোপির মাধ্যমে তাঁর পাকস্থলীতে ক্যানসার ধরা পড়ে (১নং বায়োপসি স্লাইড নং ৯৬২/৯ ডঃ এস এম ঘোষ)। বায়োপসি রিপোর্টে 'এডিনো কার্সিনোমা' উল্লেখ ছিল। এরপর সি টি স্ক্যান করে তাঁর লিভারে 'মেটাস্টেসিস' পাওয়া যায়। রোগীকে চিগুরজন ক্যানসার হসপিটালে ভর্তি করা হয় (Reg No. এস/৯২/১৬২৬) এই অবস্থায় কেমোথেরাপি প্রয়োগের কথা চিন্তা করা হয় কিন্তু রোগীর শারীরিক দুর্বলতার জন্য তা সম্ভব হয় না। অপ্রচলিত এক বিশেষ পদ্ধতিতে 'সোরিনাম' ব্যবহার করে হোমিওপ্যাথি চিকিৎসা শুরু হয়। ১৯৯২ সালের এপ্রিল মাস থেকে এই চিকিৎসা অব্যাহতি থাকে। ১৯৯৩ সালের জানুয়ারি মাসে এস এস কে এম হাসপাতালে গ্যাসট্রোস্কোপি করে দেখা যায় পাকস্থলীর দ্রুত অনেক ছোট হয়ে গেছে। তখন আবার বায়োপসি করা হয় এবং রিপোর্ট অনুযায়ী কার্সিনোমা পাওয়া যায় (স্লাইড নং ১/৩/৯২/S/৫৫৩/৯৩ ; তারিখ ২৭.১.৯৩)। এরপর একটানা হোমিওপ্যাথি চিকিৎসা চলতে থাকে। ১৯৯৩ সালের জুলাই মাসে গ্যাসট্রোস্কোপি করা হয় কিন্তু কোনও দ্রুত পাওয়া যায়নি। বায়োপসি রিপোর্টেও ক্যানসারের কোনও উল্লেখ ছিল না (স্লাইড নং এস/৪১২৭/৯৩)। শ্রীযুক্ত হুই এখনও সুস্থ আছেন এবং স্বাভাবিকভাবে চলাফেরা ও কাজকর্ম করছেন।

সারণি

মোট যোগীয় সংখ্যা	নেত্রে অত্রান্ত তথ্য	এভিনো করসিনোমা		স্কোমাসসেল কাসিনোমা		স্মল সেল কাসিনোমা		লারজ সেল কাসিনোমা		কতদিন বেঁচেছিল বা আছে				
		পুরুষ	মহিলা	পুরুষ	মহিলা	পুরুষ	মহিলা	পুরুষ	মহিলা	৬ মাস- ১ বছর	১ বছর- ২ বছর	২ বছর- ৩ বছর	৩ বছর- ৪ বছর	৪ বছরের উপরে
১২	ফুসফুস	১	৪	৪	—	২	—	১	—	১২	১০	২	১	১
৪	পাংক্রিয়াস	১	৩	—	—	—	—	—	—	৪	২	—	—	—
১০	স্টমাক	৭	৩	—	—	—	—	—	—	১০	৬	২	২	—
১৬	নারজগার্ট	১০	৬	—	—	—	—	—	—	১৬	৪	২	২	—
৬	গল-স্রাবের	৩	৩	—	—	—	—	—	—	৬	৩	—	—	—
৬	ওরাল	—	—	—	৪	—	—	—	—	৬	২	২	—	—
৫৪										৫৪	২৭	১	৫	১

উপদেষ্টা পর্যদ

অধ্যাপক সূর্যকুমার মুখার্জী, প্রাক্তন উপাচার্য
কলিকাতা বিশ্ববিদ্যালয়, সভাপতি
অধ্যাপক দেবকুমার বসু, সহ-সভাপতি, পশ্চিমবঙ্গ
রাজ্য বিজ্ঞান ও প্রযুক্তি সংসদ
অধ্যাপিকা অসীমা চ্যাটার্জী, রসায়ন বিভাগ, কলিকাতা
বিশ্ববিদ্যালয়
অধ্যাপক এম এম চক্রবর্তী, প্রাক্তন উপাচার্য, যাদবপুর
বিশ্ববিদ্যালয়
অধ্যাপক এ কে শর্মা, উদ্ভিদবিদ্যা বিভাগ, কলিকাতা
বিশ্ববিদ্যালয়
ডাঃ হরদার সিদ্ধিকী, বিশিষ্ট চিকিৎসক
অধ্যাপক তারকমোহন দাস, কৃষিবিজ্ঞান বিভাগ,
কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক এ কে বড়ুয়া, প্রাক্তন অধিকর্তা, ইণ্ডিয়ান
অ্যাসোসিয়েশন ফর দি কাল্টিভেশন্ অফ সায়েন্স
অধ্যাপক এস সি পাকড়াশি, প্রাক্তন অধিকর্তা, ইণ্ডিয়ান
ইনস্টিটিউট অফ কেমিক্যাল বায়োলজি
অধ্যাপক ভাস্করানন্দ রায়চৌধুরী, প্রাক্তন উপাচার্য,
কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক জগতজীবন ঘোষ, বিশিষ্ট শিক্ষাবিদ
অধ্যাপক পবিত্র সরকার, উপাচার্য, রবীন্দ্রভারতী
বিশ্ববিদ্যালয়
অধ্যাপক সুবোধচন্দ্র সোম, উপাচার্য, যাদবপুর
বিশ্ববিদ্যালয়
অধ্যাপক মানিকগোপাল সোম, উপাচার্য, বিধানচন্দ্র
কৃষি বিশ্ববিদ্যালয়
অধ্যাপক অশিষ কুমার রায়, উপাচার্য, কল্যাণী
বিশ্ববিদ্যালয়
অধ্যাপক মোহিতকুমার ভট্টাচার্য, উপাচার্য, বর্ধমান
বিশ্ববিদ্যালয়
অধ্যাপক সত্যনারায়ণ ঘোষ, উপাচার্য, বিদ্যাসাগর
বিশ্ববিদ্যালয়
অধ্যাপক শিশিরকুমার মুখোপাধ্যায়, উপাচার্য,
বিশ্বভারতী

সভাপতি, বিজ্ঞান জনপ্রিয়করণ ও বিজ্ঞান শিক্ষা
সভাপতি, গ্রামীণ প্রযুক্তি ও খাদ্য সংরক্ষণ
সভাপতি, শক্তি
সভাপতি, ইলেকট্রনিক্স ও শিল্পোদ্যোগ
সভাপতি, পরিবেশ
সভাপতি, খনিজ সম্পদ
সভাপতি, কৃষি, কৃষিভিত্তিক প্রযুক্তি ও পশু সম্পদ
সভাপতি, জনস্বাস্থ্য
সভাপতি, শিল্প প্রযুক্তি
সভাপতি, জল সম্পদ ব্যবস্থা
সভাপতি, দূর সংবেদ ও সাগর উন্নয়ন
সভাপতি, জৈব প্রযুক্তি
সভাপতি, অপ্রচলিত নির্মাণ সামগ্রী
অধ্যক্ষ, প্রেসিডেন্সী কলেজ
অধিকর্তা, বেঙ্গল ইঞ্জিনিয়ারিং কলেজ (ভীমড়
ইউনিভার্সিটি)
অধিকর্তা, ইণ্ডিয়ান ইনস্টিটিউট অফ কেমিক্যাল বায়োল
অধিকর্তা, ইণ্ডিয়ান অ্যাসোসিয়েশন ফর দি কাল্টিভেশ
অফ সায়েন্স
অধিকর্তা, ইণ্ডিয়ান স্ট্যাটিস্টিক্যাল ইনস্টিটিউট
অধিকর্তা, সেন্ট্রাল গ্লাস অ্যান্ড সিরামিক রিসার্চ
ইনস্টিটিউট
অধিকর্তা, ন্যাশনাল কাউন্সিল অফ সায়েন্স মিউজিয়াম
অধিকর্তা, সেন্ট্রাল মেকানিকাল ইঞ্জিনিয়ারিং রিসার্চ
ইনস্টিটিউট
অধিকর্তা, অল ইণ্ডিয়া ইনস্টিটিউট অফ হাইজিন অ্যান্ড
পাবলিক হেলথ
অধিকর্তা, জিওলজিক্যাল সার্ভে অফ ইণ্ডিয়া
অধিকর্তা, জুওলজিক্যাল সার্ভে অফ ইণ্ডিয়া
অধিকর্তা, বোটানিক্যাল সার্ভে অফ ইণ্ডিয়া
অধিকর্তা, সাহা ইনস্টিটিউট অফ নিউক্লিয়ার ফিজিক্স
অধিকর্তা, বোস ইনস্টিটিউট
অধিকর্তা, বিড়লা প্ল্যানেটোরিয়াম

অধিকর্তা, ক্যালকাটা স্কুল অফ ট্রপিক্যাল মেডিসিন
অধিকর্তা, জুট টেকনোলজিক্যাল রিসার্চ অ্যাসোসিয়েশন
অধিকর্তা, জুট এগ্রিকালচারাল রিসার্চ ইনস্টিটিউট
অধিকর্তা, ওয়েস্ট বেঙ্গল ইলেকট্রনিক ইন্ডাস্ট্রি
ডেভেলপমেন্ট কর্পোরেশন

অধিকর্তা, সত্যেন্দ্রনাথ বোস ইনস্টিটিউট অফ
ফিজিক্যাল সায়েন্সেস
অধিকর্তা, জগদীশচন্দ্র বোস ন্যাশনাল সায়েন্স ট্যালেন্ট সার্চ
অধ্যক্ষ, টেকনিক্যাল টিচার্স ট্রেনিং ইনস্টিটিউট

সংগঠন সমিতি

অধ্যাপক রথীন্দ্রনারায়ণ বসু, উপাচার্য, কলিকাতা
বিশ্ববিদ্যালয় সভাপতি
অধ্যাপক মৃণালকুমার দাশগুপ্ত, সভাপতি, পশ্চিমবঙ্গ
বিজ্ঞান ও প্রযুক্তি একাডেমী সহ-সভাপতি
অধ্যাপক দিলীপকুমার বসু, সদস্য-সচিব, পশ্চিমবঙ্গ
বিজ্ঞান ও প্রযুক্তি সংসদ সহ-সভাপতি
অধ্যাপক শংকর মুখোপাধ্যায়, বিধানচন্দ্র কৃষি
বিশ্ববিদ্যালয় সংগঠনী সম্পাদক
অধ্যাপক স্বপন প্রামাণিক, সমাজতত্ত্ব বিভাগ, কলিকাতা
বিশ্ববিদ্যালয় মুখ্য-সম্পাদক
শ্রীদীপ ভট্টাচার্য, পশ্চিমবঙ্গ বিজ্ঞান মণ্ডল
মুখ্য-সম্পাদক
ডঃ শ্যাম চক্রবর্তী, সচিব, ফ্যাকাল্টি অফ সায়েন্স,
টেকনোলজি অ্যান্ড এগ্রিকালচার, কলিকাতা বিশ্ববিদ্যালয়
কোষাধ্যক্ষ
ডিন, সায়েন্স, কলিকাতা বিশ্ববিদ্যালয়
ডিন, টেকনোলজি, কলিকাতা বিশ্ববিদ্যালয়
ডিন, মেডিসিন, কলিকাতা বিশ্ববিদ্যালয়
ডিন, সায়েন্স, কল্যাণী বিশ্ববিদ্যালয়
ডিন, সায়েন্স যাদবপুর বিশ্ববিদ্যালয়
ডিন, সায়েন্স, বর্ধমান বিশ্ববিদ্যালয়
ডিন, সায়েন্স বিদ্যাসাগর বিশ্ববিদ্যালয়
ডিন, সায়েন্স, বিশ্বভারতী
ডিন, এগ্রিকালচার, কলিকাতা বিশ্ববিদ্যালয়
ডিন, এগ্রিকালচার, বিধানচন্দ্র কৃষি বিশ্ববিদ্যালয়
ডিন, ভেটেরিনারী সায়েন্স, বিধানচন্দ্র কৃষি বিশ্ববিদ্যালয়

ডিন, ইঞ্জিনিয়ারিং অ্যান্ড টেকনোলজি, বি ই কলেজ
(ডিম্‌ড ইউনিভার্সিটি)
ডিন, ইঞ্জিনিয়ারিং, যাদবপুর বিশ্ববিদ্যালয়
ডঃ অপরাজিত বসু, সম্পাদক, বঙ্গীয় বিজ্ঞান পরিষদ
অধ্যাপক নিত্যানন্দ সাহা, চেয়ারম্যান, কলেজ সার্ভিস
কমিশন
ডঃ অশেষ লাহিড়ী, অধিকর্তা, স্টেট ওয়েস্টল্যান্ড
ডেভেলপমেন্ট বোর্ড
অধ্যাপক জয়ন্ত বসু, সাহা ইনস্টিটিউট অফ নিউক্লিয়ার
ফিজিক্স
অধ্যাপক এ এন দাঁ, রেডিও ফিজিক্স ও ইলেকট্রনিকস
বিভাগ, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক সূর্য বসু, স্কুল অফ এনার্জি স্টাডিস,
যাদবপুর বিশ্ববিদ্যালয় অধ্যাপক জে মুখার্জী, রসায়ন
বিভাগ, কল্যাণী বিশ্ববিদ্যালয়
অধ্যাপক শিশিরকুমার মুখোপাধ্যায়, কৃষিবিজ্ঞান বিভাগ,
বিশ্বভারতী
অধ্যাপক মণীশ মুখার্জী, ইলেকট্রনিক্স বিভাগ, যাদবপুর
বিশ্ববিদ্যালয়
অধ্যাপক অনিল ভট্টাচার্য, ওয়েবকুটা
ডঃ আবীরলাল মুখার্জী, বিশিষ্ট চিকিৎসক
ডঃ তপনকুমার মুখার্জী, রেজিস্ট্রার, কলিকাতা
বিশ্ববিদ্যালয়
অধ্যাপক ডি এন বোস, আই আই টি, খড়গপুর
অধ্যাপক বাসুদেব বর্মণ, কেমিক্যাল ইঞ্জিনিয়ারিং
বিভাগ, কলিকাতা বিশ্ববিদ্যালয়

প্রদর্শনী সংগ্রহস্থ উপসমিতি

অধ্যাপক অনাদিনাথ দী, কলিকাতা বিশ্ববিদ্যালয় সভাপতি
ডঃ গুরুনাথ মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়
আহ্বায়ক
ডঃ পি. হাজরা, বিজ্ঞান ও প্রযুক্তি বিভাগ, পশ্চিমবঙ্গ সরকার
শ্রীভি কুমার, গুয়েবরেডা
ডঃ প্রতিমা চট্টোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

শ্রীএস গোস্বামী, বিড়লা শিল্প ও প্রযুক্তি সংগ্রহশালা
ডঃ সুমিতা ঝা, কলিকাতা বিশ্ববিদ্যালয়
ডঃ চিত্তরঞ্জন ভট্টাচার্য্য, কলিকাতা বিশ্ববিদ্যালয়
ডঃ পার্থ মুখোপাধ্যায়, বিধানচক্র কৃষি বিশ্ববিদ্যালয়
শ্রীজগবন্ধু কোলে, কলিকাতা বিশ্ববিদ্যালয়
শ্রীস্নাতন চট্টোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

সমন্বয় উপসমিতি

অধ্যাপক দিলীপকুমার বসু, পশ্চিমবঙ্গ বিজ্ঞান ও
প্রযুক্তি সংসদ সভাপতি
অধ্যাপক অরুণবরণ বন্দ্যোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয় আহ্বায়ক
অধ্যাপক বিশ্বরঞ্জন নাগ, কলিকাতা বিশ্ববিদ্যালয়
ডঃ তরশঙ্কর বন্দ্যোপাধ্যায়, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার
শ্রীশংকর চক্রবর্তী
ডঃ সোমগুপ্ত সেনশর্মা, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার
অধ্যাপক প্রবুদ্ধনাথ রায়, কলিকাতা বিশ্ববিদ্যালয়

ডঃ শ্যামল চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক জগদীন্দ্র মণ্ডল, কলিকাতা বিশ্ববিদ্যালয়
ডঃ ভগতী বোস, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অমরনাথ চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অসিত কুমার দত্ত, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক নরেশচন্দ্র দত্ত, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অশোক কুমার মুখোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয়
অধ্যাপক অনাদিনাথ দী, কলিকাতা বিশ্ববিদ্যালয়
ডঃ গুরুনাথ মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

আতিথেয়তা ও ব্যবস্থাপনা উপসমিতি

অধ্যাপক নরেশচন্দ্র দত্ত, কলিকাতা বিশ্ববিদ্যালয়
সভাপতি
অধ্যাপক অশোককুমার মুখোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয়
আহ্বায়ক
অধ্যাপক স্বপন ভট্টাচার্য টি টি টি আই
শ্রীগোপীনাথ চট্টোপাধ্যায়, ফোসেট
শ্রীউৎপল দত্ত, পশ্চিমবঙ্গ বিজ্ঞান মঞ্চ
ডঃ অমল সংপতি, বিজ্ঞান ও প্রযুক্তি বিভাগ,

পশ্চিমবঙ্গ সরকার
শ্রীসমরজিৎ সেনগুপ্ত, কলিকাতা বিশ্ববিদ্যালয়
শ্রীকৃষ্ণকিশোর মজুমদার, কলিকাতা বিশ্ববিদ্যালয়
শ্রীমতী ইন্দ্রাণী রায়চৌধুরী, কলিকাতা বিশ্ববিদ্যালয়
শ্রীসুমিত কর, কলিকাতা বিশ্ববিদ্যালয়
শ্রীশুভব্রত বিশ্বাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীপুলকেশ দাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীঅরবিন্দ দাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীসত্যব্রত ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়

প্রদর্শনী সংগ্রহস্থ উপসমিতি

অধ্যাপক অনাদিনাথ দী, কলিকাতা বিশ্ববিদ্যালয় সভাপতি
ডঃ গুরুনাথ মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়
আহ্বায়ক
ডঃ পি. হাজরা, বিজ্ঞান ও প্রযুক্তি বিভাগ, পশ্চিমবঙ্গ সরকার
শ্রীভি কুমার, গুয়েবরেডা
ডঃ প্রতিমা চট্টোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

শ্রীএস গোস্বামী, বিড়লা শিল্প ও প্রযুক্তি সংগ্রহশালা
ডঃ সুমিতা বার্মা, কলিকাতা বিশ্ববিদ্যালয়
ডঃ চিত্তরঞ্জন ভট্টাচার্য্য, কলিকাতা বিশ্ববিদ্যালয়
ডঃ পার্থ মুখোপাধ্যায়, বিধানচল্ল কৃষি বিশ্ববিদ্যালয়
শ্রীজগবন্ধু কোলে, কলিকাতা বিশ্ববিদ্যালয়
শ্রীস্নাতন চট্টোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

সমন্বয় উপসমিতি

অধ্যাপক দিলীপকুমার বসু, পশ্চিমবঙ্গ বিজ্ঞান ও
প্রযুক্তি সংসদ সভাপতি
অধ্যাপক অরুণবরণ বন্দ্যোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয় আহ্বায়ক
অধ্যাপক বিশ্বরঞ্জন নাগ, কলিকাতা বিশ্ববিদ্যালয়
ডঃ তারাকান্ত বন্দ্যোপাধ্যায়, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার
শ্রীশংকর চক্রবর্তী
ডঃ সোমেন্দ্র সেনশর্মা, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার
অধ্যাপক প্রবুদ্ধনাথ রায়, কলিকাতা বিশ্ববিদ্যালয়

ডঃ শ্যামল চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক জগদীন্দ্র মণ্ডল, কলিকাতা বিশ্ববিদ্যালয়
ডঃ ভগতী বোস, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অমরনাথ চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অসিত কুমার দত্ত, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক নরেশচন্দ্র দত্ত, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক অশোক কুমার মুখোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয়
অধ্যাপক অনাদিনাথ দী, কলিকাতা বিশ্ববিদ্যালয়
ডঃ গুরুনাথ মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

আতিথেয়তা ও ব্যবস্থাপনা উপসমিতি

অধ্যাপক নরেশচন্দ্র দত্ত, কলিকাতা বিশ্ববিদ্যালয়
সভাপতি
অধ্যাপক অশোককুমার মুখোপাধ্যায়, কলিকাতা
বিশ্ববিদ্যালয়
আহ্বায়ক
অধ্যাপক স্বপন ভট্টাচার্য টি টি টি আই
শ্রীগোপীনাথ চট্টোপাধ্যায়, ফোসেট
শ্রীউৎপল দত্ত, পশ্চিমবঙ্গ বিজ্ঞান মঞ্চ
ডঃ অমল সংপতি, বিজ্ঞান ও প্রযুক্তি বিভাগ,

পশ্চিমবঙ্গ সরকার
শ্রীসমরজিৎ সেনগুপ্ত, কলিকাতা বিশ্ববিদ্যালয়
শ্রীকৃষ্ণকিশোর মজুমদার, কলিকাতা বিশ্ববিদ্যালয়
শ্রীমতী ইন্দ্রাণী রায়চৌধুরী, কলিকাতা বিশ্ববিদ্যালয়
শ্রীসুমিত কব, কলিকাতা বিশ্ববিদ্যালয়
শ্রীশুভ্রত বিশ্বাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীপুলকেশ দাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীঅরবিন্দ দাস, কলিকাতা বিশ্ববিদ্যালয়
শ্রীসত্যব্রত ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়

প্রকাশক উপসমিতি

শ্রীশংকর চক্রবর্তী সভাপতি
ডঃ সোমগুপ্ত সেনশর্মা, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার আহ্বায়ক
শ্রীডি ভট্টাচার্য, বিজ্ঞান ও প্রযুক্তি বিভাগ, পশ্চিমবঙ্গ
সরকার

ডঃ অনীশ দেব, কলিকাতা বিশ্ববিদ্যালয়
শ্রীপ্রদীপ ঘোষ, কলিকাতা বিশ্ববিদ্যালয়
ডঃ পীযুষকান্তি সাহা, কলিকাতা বিশ্ববিদ্যালয়
শ্রীমানস সান্যাল, ফোসেট
শ্রীসমর বাগচী

গবেষণাপত্র বাছাই উপসমিতি

অধ্যাপক বিশ্বরঞ্জন নাগ, কলিকাতা বিশ্ববিদ্যালয়
সভাপতি
ডঃ তারাকঙ্কর বন্দ্যোপাধ্যায়, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার আহ্বায়ক
ডঃ শ্যামল চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয়
অধ্যাপক নিত্যানন্দ সাহা, কলেজ সার্ভিস কমিশন
ডঃ শুভ্রত সেনগুপ্ত, ইণ্ডিয়ান ইনস্টিটিউট অফ
কেমিক্যাল বায়োলজি
ডঃ অমিয়কুমার হাটী, স্কুল অফ ট্রপিক্যাল মেডিসিন

ডঃ সুবিমল সেন, সাহা ইনস্টিটিউট অফ নিউক্লিয়ার
ফিজিক্স
অধ্যাপক বুদ্ধদেব ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়
ডঃ অজয় অধিকারী, ইণ্ডিয়ান স্ট্যাটিস্টিক্যাল
ইনস্টিটিউট
অধ্যাপক আর কে মণ্ডল, বসু বিজ্ঞান মন্দির
অধ্যাপক গৌরীপদ দত্ত
শ্রী শৌলপতি গুপ্ত
অধ্যাপক এ কে সাহা, অধিকর্তা ডব্লু বি আর ই ডি এ

অর্থসংক্রান্ত উপসমিতি

অধ্যাপক প্রবুদ্ধনাথ রায়, কলিকাতা বিশ্ববিদ্যালয়
সভাপতি
ডঃ শ্যামল চক্রবর্তী, কলিকাতা বিশ্ববিদ্যালয় আহ্বায়ক
অধ্যাপক রঞ্জুগোপাল মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়
শ্রীএস বি মাহাতো, পশ্চিমবঙ্গ রাজ্য বিজ্ঞান ও
প্রযুক্তি সংসদ
শ্রী এ কে মাকর, বিজ্ঞান ও প্রযুক্তি বিভাগ, পশ্চিমবঙ্গ
সরকার
শ্রীসোমনাথ ভট্টাচার্য, পি বি ডি এম
শ্রীসুদীপ্তি বন্দ্যোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়
সংবাদ ও প্রচার সংক্রান্ত উপসমিতি
অধ্যাপক জগদীন্দ্র মণ্ডল, কলিকাতা বিশ্ববিদ্যালয়
সভাপতি
ডঃ তপতী বোস, কলিকাতা বিশ্ববিদ্যালয় আহ্বায়ক

ডঃ মেহাংগুকুমার দাশগুপ্ত, কলিকাতা বিশ্ববিদ্যালয়
শ্রীদুলাল মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়
ডঃ অলোক সেন, আকাশবাণী
শ্রীসুভাষ সান্যাল, দূরদর্শন
শ্রীযুগলকান্তি রায়
শ্রীরতনমোহন খান, বঙ্গীয় বিজ্ঞান পরিষদ
শ্রীভবতোষ ভট্টাচার্য
শ্রীতপন সাহা, পশ্চিমবঙ্গ বিজ্ঞান মঞ্চ
ডঃ পার্থ বন্দ্যোপাধ্যায়, এন সি এস এম
শ্রীঅর্চন মজুমদার, ফোসেট
শ্রীএস কে দাশগুপ্ত, বিজ্ঞান ও প্রযুক্তি বিভাগ,
পশ্চিমবঙ্গ সরকার
শ্রীরোহিত বসু, অধিকর্তা, নন্দন
ডঃ অজিতকুমার বেরা, কলিকাতা বিশ্ববিদ্যালয়

সহযোগী

শ্রীমতী চন্দনা আদিত্য, কলিকাতা বিশ্ববিদ্যালয়

শ্রীসত্য দাশ, কলিকাতা বিশ্ববিদ্যালয়

শ্রীতাপসকুমার মাইতি, কলিকাতা বিশ্ববিদ্যালয়

শ্রীগুরুপ্রসাদ মহাস্তি, কলিকাতা বিশ্ববিদ্যালয়

শ্রীআশিস সরকার, কলিকাতা বিশ্ববিদ্যালয়

শ্রীমতী সোনারেখা মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

শ্রীবসুমন ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়

শ্রীসৌমিক ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়

শ্রী সুমন ভট্টাচার্য, কলিকাতা বিশ্ববিদ্যালয়

শ্রীমতী মেঘমিতা ঘোষ, কলিকাতা বিশ্ববিদ্যালয়

শ্রীপঙ্কজ পোদ্দার, কলিকাতা বিশ্ববিদ্যালয়

শ্রীউজ্জ্বল মুখোপাধ্যায়, কলিকাতা বিশ্ববিদ্যালয়

শ্রীমতী জয়াতী সাহা, কলিকাতা বিশ্ববিদ্যালয়

শ্রীমতী অর্পিতা বল, কলিকাতা বিশ্ববিদ্যালয়

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(OTHER THAN THOSE IN THE BULLETIN OF THE CALCUTTA SCHOOL OF TROPICAL MEDICINE)

For omissions please see Note by compiling editor. We shall be grateful if omissions are pointed out to enable inclusion in the Addendum.

For abbreviations used, see list at the end.

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Errata : 369 & 387, 646 & 647 are repetitions

CRITICAL OVERCOME EPISODE 2

In the year 1999, another eventful thing happened in Delhi. I was invited at a prestigious seminar on Oral Oncology organized by the International Congress on Oral Oncology to present a paper. This I was able to present the abstract of the paper with the kind support of Dr. A. K. Varma. The entire paper was later published in the book titled 'Oral Oncology Volume VI' and soon after that a few more papers also got published. I have already shared three of those papers at the beginning of this book titled 'The Management of Cancer in totality - India can take a Lead – In Theory and Application', 'Psorinum Makes a Major Break Through in the Treatment of Tobacco Related Lung Cancer' and 'Non-conventional Treatment of Tobacco Related Cancer Gradually Gets Right Perspective through Psorinum Therapy. However, the first paper that got published in the 'Oral Oncology Volume VI' I am presenting it here.

Throughout my life I had to fight a long battle. Before 1995 my work had fallen into a loop of complexities and legalities of authentications. I was battling harder but not finding a way out. After 1999 I found Dr. A. K. Varma standing by my side and he helped me to overcome certain situations. He documented many of my works and these documentations paved a way for me by creating somewhat a legal platform.

Again since 2006 Dr. Aradeep Chatterjee has structured and developed this work and tried to build it a more comprehensive and methodical way and on many occasions presented several papers on global scientific forums. Gradually our facility-centre got membership from ASCO as a cancer research centre and many international journals got published under name of the Critical Cancer Management Research Centre and Clinic. I desire to throw more light on this subject in the next volume.



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ABSTRACTS



International Union Against Cancer



National Institute of Dental Research



International Association for Dental Research

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USE OF PSORINUM IN THE TREATMENT OF CANCER

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Tumor invasion, metastasis and resistance to chemotherapeutic drugs or radiation are major obstacles for the successful treatment of cancer. This refractoriness of solid tumors to cytotoxic therapies has led to the exploration of new therapeutic modalities and strategies and a regimen which aims to increase the specificities and efficacy and reduce the toxicity of the anticancer drugs.

Good response was obtained when psorinum was orally administered for the treatment of the terminal cancer patients (ECOG 3 and 4). Out of the 275 patients treated for carcinoma of various organs viz. oral, head & neck, lung, stomach, pancreas, gall bladder, liver etc., the preliminary results are quite encouraging and interesting. About 80 percent of the patient was found responding to this therapy. During the first 6 month of the therapy most of the cases showed gastrointestinal and hepatobiliary symptoms along with improvement in liver function. Haematological picture also indicated satisfactory progress. Along with the psorinum therapy, supportive treatment like blood transfusion, abdominal or pleural paracentesis, analgesic, bronchodilators and stenting of hepato-pancreato-biliary system etc., done as and when required. Significant improvement in ECOG score was obtained, quality of life was improved and life prolonged in most of the cases. Total disappearance of tumor were seen in 45 patients out of which few were oral cancer cases.

The medicine was administered orally and so far no adverse side effects were noticed in any case. Before the start of the therapy informed consent was taken from all the patients.

Though the exact mode of action of Psorinum in regression of tumours is still not known, however, the results suggest that the drug could be a non specific immunomodulator. Biological response modifiers (BRM) with antitumour immunopotential activity, derived from plant, microbial organism, bacterial cell fraction, cytokines and thymic humoral fractions are known to play an important role in cancer immunotherapy. OK-432 (Whole cell preparation from *Streptococcus pyogenes*), PSK (the crude glycoprotein fraction of *coriolus versicolor*), Lentinam (*B*-glucan purified from *schizophyllum commune fries*) are now widely used in Japan. The *B*-glucan moiety is the common active site in the later three BRM.

ORAL ONCOLOGY



VOLUME VI

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Use of Psorinum in the Treatment of Cancer

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INTRODUCTION

Oral carcinoma is relatively uncommon in most parts of the world. However, in India, it occupies the top rank among male cancers and ranks within the first 5 among female cancer. Oral cancers of India are often linked to the tobacco chewing habit (Gupta, 1993).

Tumour invasion, metastasis and resistance to chemotherapeutic drugs or radiation are major obstacles for the successful treatment of cancer. This refractoriness of many solid tumours to cytotoxic chemotherapy has led to the exploration of new therapeutic modalities viz. immunotherapy, gene therapy, anti angiogenesis, tumour vaccinations, biological therapy, development of resistance modifying agent (RMA) and many forms of alternative & complementary cancer therapies all over the world. Immunotherapy does not have a direct cytotoxic effect on the cancer cell but is an attempt to promote rejection of the tumour by the host, chiefly through the cellular arm of the immune system (Quan and Palackdharry 1997). Anti angiogenesis drugs are directed to block the tumour blood vessels through which the tumour get the oxygen and nutrients. Gene therapy aims to replace the defective gene or block the oncogenes. In most the alternative & complementary cancer therapies the mode of action are not exactly known.

Complementary and alternative medicine can be defined as those medicine system, practices, interventions and applications that currently are not part of the dominant or conventional medical system. There are more than 300 topics under the term complementary and alternative medicine that can be divided into seven major categories on the basis of philosophy, approach to the patient, and orientation.

The present study was initiated to see the effect of a conventional drug Psorinum in terminal cancer patients in a non conventional regimen.

MATERIALS & METHODS

Drug : The drug Psorinum is the alcoholic extract of the scabies scrub, slough and pus cells. The drug was administered orally, approx. 0.01 ml/kg body weight per day as a single dose in empty stomach.

Patients: 275 cases suffering from carcinoma of different organs in stage III & IV and performance status (ECOG) of 3 or 4 were taken for the study as a

randomized open trial, after informed consent had been obtained from them.
Investigation : Prior to the therapy, all the diagnosed patients were evaluated and staged by routine and special diagnostic techniques including skiagraph, barium meal, fibroptic endoscopy, ultrasonography, whole body scan and detailed hemogram. The histopathology was reexamined in all the detected cases carcinoma and each case irrespective of clinical stage was evaluated by oncologist whether or not was treated previously by surgery, radio and/or chemotherapy, either alone or in combination according to the merit of the case. In most of the cases conventional therapy was not recommended by oncologist considering poor prognosis and bad treatment outcome. Few were treatment failure cases.

Management : Along with the Psorinum therapy, symptomatic therapy for control of infection, bleeding and nutritional deficiencies were also given. Blood transfusion, abdominal or pleural paracentesis, analgesic, bronchodilators and stenting of the hepato-pancreato-biliary system and palliative relevant surgeries were done as and when required.

Assessment : Tumour size shrinkage was regularly assessed by physical examination, skiagraph, roentgenogram, sonography or CT scanning as applicable. CT scan was preferred for accurate evaluation of size.

RESULTS

Good response was obtained when psorinum was administered for the treatment of the terminal cancer patients (ECOG 3 and 4). Out of the 275 patients treated for carcinoma of various organs viz. oral, head & neck, lung, stomach, pancreas, gall bladder, liver etc., the preliminary results are quite encouraging and interesting. In total 25 oral/head&neck cases were studied. About 80 percent of the patient was found responding to this therapy. During the first 6 month of the therapy most of the cases showed marked remission of presenting symptoms like pain, respiratory, gastrointestinal and hepatobiliary symptoms along with improvement in liver function. Haematological picture also indicated satisfactory progress. Along with the psorinum therapy, supportive treatment like blood transfusion, abdominal or pleural paracentesis, analgesic, bronchodilators and stenting of hepato-pancreato-biliary system etc., palliative and relevant surgeries viz. debulking and bypass were done as and when required. Significant improvement in ECOG score was obtained, quality of life was improved and life prolonged in most of the cases.

During the first year, reduction of tumour size more than 70% of the original size were observed in 10 percent of cases. Reduction of approx. 50 percent of the tumour size were observed in 16 percent of cases. In few cases it was observed that the performance status have improved despite very little regression of tumour growth. In 20 percent of cases there were insignificant reduction in size of tumour with waxing and waning of clinical features indicating fluctuating performance status. As many as 13.2 percent of cases died either from infection, bleeding episodes or organ failure.

Complete disappearance of tumour were observed in 45 cases viz. 3 oral, 11 lung, 15 stomach, 7 pancreas, 5 gall bladder, 1 corectal and 3 liver. No malignant cells were detected histopathologically from the primary site in those cases. Median survival for complete responders was 34 months compared

with 9 months for partial responders and 4 months for non responders. The mean survival months for lung, stomach, pancreas, gall bladder, liver and colorectal were 17.0, 22.5, 14.9, 13.0, 7.9 and 30.9 months respectively.

In the patients where the tumour disappeared completely, had a good disease free survival record. And so far no adverse side effects were noticed in any cases.

DISCUSSION

When the trial started the drug psorinum was administered with out any supportive care, hence, though there was considerable shrinkage as far the tumours was concerned but the mortality rate remained very high. Later when the supportive treatment was tagged viz. blood transfusion, abdominal or pleural paracentesis, analgesic, bronchodilators and stenting of the hepatopancreato-biliary system and palliative relevant surgeries etc., there was considerable decline in the mortality rate.

Few cases of cancer of various organs were studied at first, when it was found that psorinum was working good as a anti-cancer drug along with the supportive care (Chatterjee et al.1995). Few oral cancer were also treated initially with this drug and the results were good. It was then decided that it would be interesting to see the effect of this drug in such cancers where the prognosis is very poor with the conventional treatment and hence, much of the attention was diverted for the cancer of pancreas, liver, lungs, stomach etc.

Researcher both in the United States and other western countries suggest that significant number of people are involved with various form of alternative medicine. However, the reasons for such use are, at present poorly understood. Grothey et al. (1998) reports number of patients treated with conventional oncological regimens also use alternative medicine, most of them because of a polypragmatic attitude to tumour treatment. Alternative medicine was used largely as complementary and not an alternative to conventional medicine. However, in India the scenario is quite different the problem do not only relates to economic conditions but are specific and have very serious implication on the course of treatment. Most of the patient are on two or more therapies at a given time. Despite knowledge patient will present in advance stage, decision making regarding further treatment rest with the family and not with the patient. Opinion of elders in family, are more important than that of a qualified specialist. There is a natural belief of non responsiveness of malignancy, allowing psychologically the relatives to take decisions against treatment, thus making it impossible to run a good trial (Pendharkar 1998). These problems are common for all the tropical countries and do not have relationship to level of education and financial status.

Though the exact mode of action of Psorinum in regression of tumours is still not known, however, the results suggest that the drug could be a non specific immunomodulator. Biological response modifiers (BRM) with antitumour immunopotential activity, derived from plant, microbial organism, bacterial cell fraction, cytokines and thymic humoral fractions are known to play an important role in cancer immunotherapy. OK-432 (Whole cell preparation

from *Streptococcus pyogenes*), PSK (the crude glycoprotein fraction of *coriolus versicolor*), Lentinan (β -glucan purified from *schizophyllum commune* fries) are now widely used in Japan. The β -glucan moiety is the common active site in the later three BFM (Tanaka et al. 1998).

CONCLUSION

The Psorinum therapy is quite promising for the treatment of cancer of lungs, stomach, pancreas, gall bladder, liver and colon and is free from any side effects. This therapy could also be effective in oral cancer. It could be safely given to patient of older age groups viz. 60 years and above, where the treatment outcome and prognosis is very poor with the conventional therapy. Detailed investigation is needed to know how and why the entire tumour is slowly disappearing in few cases. In cost effective basis this therapy is very inexpensive and above all the drug is administered orally. Future studies with larger number of cases are needed to assess the exact effectiveness of this therapy. Since there is little information on the long term toxicity and pharmacokinetics of this treatment, detailed investigation are to be taken up in this direction.

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Oncology



Nucleoside analogues in cancer therapy
Management of thyroid cancer
Brain-tumour drug resistance



Reflection & Reaction

Use of alternative cancer medicine in India

The use of complementary and alternative medicine (CAM) by cancer patients is becoming widespread. This is a reflection of the many needs and concerns that are currently not being met by conventional medical practice. Significant proportions of cancer patients in developed countries use complementary therapies as adjuncts to conventional symptom management to improve their quality of life.¹ However, the situation in less-developed countries such as India, is quite different. Around 80% of cancer patients have late stage incurable disease when first diagnosed.² This not only complicates the treatment options, but also makes palliation difficult. Procurement of oral morphine for the treatment of pain in terminal cancer patients is another problem because of cumbersome legislation.³ In remote parts of the country, patients have limited access to medical services and many are compelled to try alternative medicines, including naturopathy, biopathy, homeopathy, home remedies, wheat-grass therapy, hydrotherapy, acupuncture, auricular therapy, osteopathy, and vipasana.⁴

About 70% of the Indian population obtain medical help from private physicians, and nearly half of those seek help from alternative or

traditional medical practitioners. Appalling poverty and hygiene, and a complex social dynamic, pose major hurdles in treatment efficacy.⁵ Cancer patients often report to clinics when their malignancy has reached an advanced stage. It is possible that many factors, such as ignorance, socio-economics, poor roads and transportation, lack of communication facilities, inadequate medical facilities in the primary healthcare sector, and poor infrastructure, all contribute to this situation. For example, a recent estimate has shown that there is a shortfall of about 750 teletherapy machines.⁶ Since a majority of the population is not covered by insurance, financial constraints become a major obstacle for many patients in their fight against cancer.

Great advances have been made in the treatment of some tumours and new advances in surgery, radiotherapy, and chemotherapy has lead to an increase in cure rates—but at a price beyond the reach of many cancer patients living in the developing world.⁷

There are limited studies on the use of CAM by Indian cancer patients. In Kolkata, I interviewed 200 tissue-biopsy-confirmed cancer patients, or their caregivers, on why they were trying an alternative cancer therapy

called Psorinum. Since the publication of an anecdotal report alleging improved survival among many people with advanced-stage cancer,⁸ both the public and many oncologists now regard this approach as effective. This unconventional treatment comprises a combination of homeopathy and natural medicines along with conventional supportive care. The responses to the survey are shown in table 1. In general, many patients claimed that they were trying Psorinum because there was either no other option or because of financial constraint. The survey showed that 118 men (59%) and 82 (41%) women were trying the therapy and of these, 85% came from urban communities and 15% came from rural areas. Most patients had gastrointestinal cancer (57%), while 21% had lung cancer, 12% had carcinoma of another major organ, and 10% had other cancer types. Furthermore, and strikingly, nearly 60% of the patients were over 60 years of age.⁹

Other popular alternative medicines used in India for cancer treatment include Ayurveda, and herbal, natural, tribal, and folk medicines. The Ayurvedic medicines, Valipani, Navjeevan, and Kamdudha, have shown efficacy in some leukemia patients.¹⁰ In addition, another

Table 1. Patient response when asked why they were using an alternative "anticancer" medicine

Rank	Category	Response	Number of patients (n=200)
1	No treatment options	Advice of oncologist No specific treatment recommended by oncologist Not responding to conventional treatment	40 (20%)
2	Financial	Financial and economic problems No health insurance Treatment in private clinic too costly Already invested a considerable amount in conventional treatment	32 (16%)
3	Quality of life	Patient too weak to undergo or continue chemotherapy Too old for conventional treatment Adverse side-effects to radiotherapy	30 (15%)
4	Advertisement	Advice of a friend who used a certain therapy Read in newspaper	25 (12.5%)
5	Pain management	Like to use this therapy primarily for palliation	23 (11.5%)
6	Patient management	Live near the clinic and easy to manage the patient at home Availability of doctor at all times of the day and night	15 (7.5%)
7	Multimodal therapy	Like to use alternative medicine along with the conventional treatment	14 (7%)
8	Frustration	Not convinced with oncologist's advice Need further information about cancer and options	10 (5%)
9	Faith	Belief that homeopathic medicine can cure cancer	8 (4%)
10	Experimental	Suffering from pancreatic cancer; would like to try an experimental therapy	3 (1.5%)

Ayurvedic formulation, Maharishi Amrit Kalash, is proving to be effective in controlling the side-effects of chemotherapy.

Until there is a dramatic improvement in cancer mortality using conventional treatments, CAM will continue to attract many cancer patients. With the high propensity for late-stage diagnosis, many treatments offer little more than palliative care, and it is possible that CAM approaches will play an important role in those situations when cure is no longer a realistic objective.

The findings of the survey reported here suggest that we should have an open mind about the use of CAM. About 50% of the world's cancer burden is carried by developing

countries that, ironically, have access to only 5% of the resources available to fight the disease. In the developing world, and arguably the developed world, CAM may become an important component of modern oncology if integrated properly in to mainstream medicine.

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Cystic Fibrosis heterozygosity: Darwinian bet on cancer protection?

In the April issue of *The Lancet Oncology*, Mel Greaves proposed a neo-Darwinian viewpoint on cancer causation.¹ The author suggested that the current risk for skin, breast, and prostate cancer might be enhanced by some of our ancestral adaptive advantages related to migration or reproduction. He admits, however, that there is limited genetic proof to fully endorse this theory in humans.

Although controversial, inherited advantage represents the most solid explanation² for the high incidence (about 5% in Caucasian populations) of heterozygous mutations in the cystic fibrosis (CF) gene; a somatic gene that blocks the epithelial-cell ion channels when mutated and leads to multiorgan dysfunction in homozygotes. Theories of selection-induced CF mutations causing protection against bacterial diseases have been classically supported, but also reproductive advantages have been suggested among subgroups of heterozygote carriers.³

The possibility of an inverse relationship between CF gene mutations and cancer incidence was first reported among a Welsh population, in which a lower than expected rate of certain tumours was found for CF carriers.⁴ Additional analyses showed no increased incidence of any cancer in the carrier group, while lower risks for

certain malignancies, such as melanoma, were confirmed in heterozygote individuals.⁵

This data, and that of Neglia and co-workers⁶ who found no increase in the overall cancer incidence among CF homozygotes despite higher rates of digestive-tract tumours, stimulated an experimental study⁷ that reported a stronger inhibition of human breast-cancer proliferation in mice with 1 or 2 copies of the mutated CF gene. It was suggested that ion-channel blockades, leading to high concentrations of extracellular ATP, caused the growth inhibition. Likewise, overlapping pathways have also been used to explain the reduced invasiveness seen in human prostate cancer-cells in which ion channels were blocked pharmacologically.⁸

We agree that competitive procreation may be the primary endpoint of evolution, but it is hard to justify that human evolution is trying a fecundity advantage at any price, if this means the perpetuation of risky genotypes that may cause increased cancer incidence in future generations. If we speculate based on CF heterozygosity as the best model of adaptive mutation in white populations to date, then current data, showing no increased risk of cancer among CF carriers, are pointing away from the hypothesis proposed by Greaves.^{4,9} Further population-based

and experimental evidence is now needed to address the intriguing possibility that CF heterozygosity may prevent melanoma, breast, or prostate cancer.

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ATTITUDES OF PATIENTS TO ALTERNATIVE MEDICINE FOR CANCER TREATMENT

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Abstract

Awareness of attitudes to different types of medicine is very important for establishment of cancer prevention programs. Alternative medicine has become an important feature of oncology regardless of geographic region, and in India, the majority of cancer patients present at late advance stage of disease when curative treatment cannot be initiated. Given the lack of facilities it is no surprise that many Indian cancer patients try various complementary and alternative medicines, despite the fact that little is known about their therapeutic efficacy and toxicity. A study was conducted in 300 biopsy proven cancer patients undergoing alternative cancer therapy with Psorinum in Kolkata. The main aim of the study was to analyze the patients' / caregivers narratives regarding the therapy they have been trying. One hundred and ninety five patients (65%) have consulted their oncologists before trying this therapy. About 18.5% of the patients expressed satisfaction with the therapy due to the holistic nature and team approach employed for patient management. The cost of the therapy was within the reach of many cancer patients belonging to the underprivileged segment of the society, contributing to its immense popularity in Kolkata. Whether this can be translated into a willingness to use similar natural compounds for cancer prevention and treatment purposes now needs to be analysed.

Key Words : Awareness – alternative cancer therapy – homeopathy – India

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Introduction:

Complementary and Alternative medicine (CAM) is a highly visible part of contemporary health care and has got an important role in palliative cancer care (Pal and Mittal, 2003). No longer restricted to the lay sector and the medical fringe, such practices can be found in mainstream hospitals and cancer centres (Pal, 2002a). In the United States and in other more developed countries many millions of patients spend billion of dollars each year on complementary and alternative medicine (Vicker and Cassileth, 2001). Many alternative cancer therapies like antineoplastons, hydrazine sulphate, shark cartilage, Cancell, Coenzyme Q 10, 714X are very popular in USA (Schraub, 2000; Cassileth and Chapman, 1996; Shukla and Pal, 2004). Immunoargumentative therapy in Bahamas, Essaic in Canada, Mistletoe in Europe, Chinese herbs in China and Ayurveda in India are all very popular alternatives for cancer treatment. Proposal of 'miracle' cancer therapy without scientific evidence of effectiveness have been advocated numerous times in many different countries some of these have found the enthusiastic support of the local public and press (Lemer and Kennedy, 1992). One such example the Di Bella multi-therapy generated intense public interest in Italy few years ago (Pellegrini, 1998). Two Indian alternative cancer therapies 'Antineoplastin' and 'Methyl gloxal' also created a great deal of public interest in Kolkata (Pal, 2001).

There is widespread frustration among cancer patients concerning conventional medicine's inability to treat many cancers effectively. In the absence of real treatment gains for the majority of cancer and chemotherapy's side effects have become increasingly intolerable to many (Cassileth and Chapman, 1996). Focus on 'natural products' for gentler as well as more effective substitute for standard cancer treatment is a top priority now. In less developed countries majority cancer patients have less access to mainstream cancer treatment, many patients with cancer who live in these countries are never seen in a hospital (Sansom and Mutuma, 2002). Conventional care is rarely free and almost always beyond the reach of many. Lack of proper infrastructure (Sharma, 2001), ignorance (Chaturvedi et al., 2002) and above all absence of social security schemes plays an important role for selecting alternative cancer therapies that are replacement for the conventional treatment (Sureshkumar and Rahagopal, 1996). Very few scientific reports are available on the different alternative cancer therapies that are tried by the Indian patients. The present investigation was undertaken to access viewpoints on a popular alternative cancer therapy called Psorinum in northern Kolkata.

Patients and Methods

Settings

The present investigation was conducted in the clinic of first author situated in northern Kolkata.

Patients

The sample comprised of 300 tissue biopsy diagnosed cancer patients (179 men and 121 women ranging from 18 to 92 years, mean age 59+- 11.98 years).

Inclusion & Exclusion Criteria for patients who wanted to try this therapy, but the therapy was offered to those for whom conventional treatment was not recommended by oncologists considering poor prognosis and doubtful treatment outcome. Some of the patients were treatment failure case. Informed consent either verbal or signed was taken from each patient before starting the therapy.

Interview

Detailed interview was conducted in 300 and 253 patients and/or their caregivers at the start and at the stoppage / 6 months after start of the therapy, respectively. The interview was aimed to record the feelings of the patients regarding the alternative cancer therapy.

Monitoring Efficacy

The therapeutic efficacy of this alternative cancer therapy was assessed by the comparative study of tumour / lesion size shrinkage. Decrease in tumour size, cancer related pain, drying-up of pleural effusion and ascites were considered as response. The changes in tumour / lesion size were monitored by roentgenogram, endoscopy, ultrasonography, nuclear scan or CT scan as applicable, after every 3-4 months.

Results

One of the major reasons cited by patients for opting for this alternative therapy was no treatment options for treatment because of advance stage of the disease (22%), followed by economic problems (16%) and to improve quality of life (13%). About 11.6% of the patients were drawn to this therapy because of advertisement. Twenty nine (9.6%) patients wanted to try this therapy mainly for palliation. The other reasons stated by patients are illustrated in Figure 1.

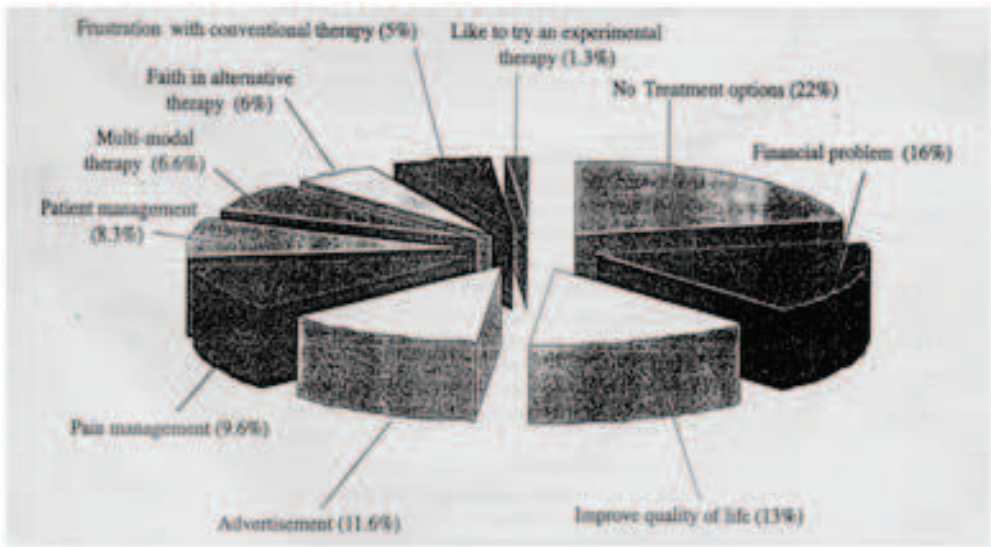


Figure1. Patient Responses When Asked Why they Want To Trying Psorinum Therapy (N= 300)

Table: The details of the patients who have undergone the Psorinum therapy

Total number of patient interested to try this therapy	300
Patient who could not take the therapy due to co-morbid problem / or Died before the therapy could be initiated	15
Patient in early stage of malignancy referred to conventional medicine	10
Total number of patients actually tried this therapy	275
No. of patients received chemo or radiotherapy before starting therapy	47
No. of patients received complete cycles of chemo / radiotherapy	25
No. Of patients received incomplete cycles of chemo / radiotherapy	22
Patients who dropped out / lost to follow-up	22
Patients using other CAM or Chemotherapy along with Psorinum therapy	15
Patients where marked tumour regression was observed	45
Patients who had mild side effects with this therapy	12

The views of the patients and / or their caregivers who tried this alternative cancer therapy is given in Table 2. One hundred and fifty six (52%) patients were from Kolkata, 65 (21.6%) were from other major cites of the state of West Bengal, and 39 (13%) patients came from small townships and villages. Few patients 25 (8.3%) were from the neighbouring country Bangladesh and 15 (5%) were from other states of India. A notable feature was the number of old patients, 159 (57.8%) were either 60 years or more. One hundred and ninety five (65%) patients consulted their oncologists before coming forward to try this alternative therapy. Majority of the cancer patient trying this alternative therapy were in palliative (64.7%) and terminal (31.6%) condition respectively. The Psorinum therapy did not have any toxic side effects, although 12 (4.3%) patients complained of mild oral irritation, skin allergy and gastritis. Around 37.9% of the patients (96/253) who took this therapy either had stroke or died of stroke related incidence.

Table 2: Patients views who had tried 'Psorinum' Therapy

Rank	Category	Response	No. of Patients (N=253)
1.	Managemant	I like the holistic approach employed for patient management Treatment was possible while staying in home Blood transfusion, tapping of ascites, pleural paracentesis was possible with out living my home Hospital visit less often.	47 (18.5%)
2.	Belief	After mark remission of my disease I believe that this alternative therapy works for cancer treatment My patient had a remarkable improvement and complete shrinkage of tumour with this therapy.	42 (16.6%)
3.	Frustration	I was not given any guaranty regarding cancer cure This therapy is noteffective in all types of cancer This therapy does not work in all terminal patients We tried this therapy because of financial problems.	33 (13%)
4.	Team Approach	Though the therapy seemed ineffective in our case but we continued it. Because of the team effort of the doctor to manage our problems	26 (10.2%)
5.	Guidance & Reference	The doctors have guided us in right direction. The doctors helped and guided us in taking important decision. Due to the reference of Dr. Chatterjee we could get an early appointment in hospitals for checkup.	25 (9.8%)
6.	Economic	The 'Psorinum' therapy was quite cost-effective and promising 'Psorinum' therapy was quite inexpensive and effective in my case	21 (8.3%)
7.	Litening to patient	The doctor was ready for any discussion on cancer treatment We felt more comfortable discussing any topic on cancer with him The doctor gave more time listening to patient's problem	20 (7.9%)
8.	No side effects	The therapy is not having any side effects No immediate toxicity was observed. No loss of hair.	18 (7.1%)

9.	Pain	The therapy was effective in controlling of cancer related pain	16
10.	Continuation	My tumour disappeared following 'Psorinum' therapy but reappeared after stoppage of the therapy I have to restart therapy The therapy has to be taken for a very longer period	5 (1.9%)

Discussion

Interest in alternative therapies is growing rapidly (Ernst, 2003). Some from of herbal therapy to treat cancer is popular throughout the world (Smith and Boon, 1999; Cassileth and Chapman, 1996). Natural products from plant are rich sources used for treating a number of diseases. In the field of anti-cancer therapy many active cytotoxic agent were originally developed from natural sources (Schwartzmann et al., 2002). Examples include etoposide from *Podophyllum peltatum*, vincristine from *Catharanthus roseus*, and gemcitabine from *Crypthotheca crypta* and Paclitaxel from *Taxus brevifolia*. Traditional drugs have been the starting point for the discovery of many important drugs. Most of the herbal drugs are a mixture of a number of plant ingredients. The cumulative effect increase s the efficacy of the drug in curing the disease (Palani et al., 1999). Essiac one of the most popular herbal alternatives in North America is also derived from 4 herbs (Kaegi, 1998). Iscador, a derivative of mistletoe, is a popular cancer remedy in Europe, has been used as folk treatment for centuries (Cassileth and Chapman, 1996). The homeopathic mother tinctures used in the Psorinum therapy contained alcoholic extract of various medicinal herbs viz. *Chelidonium Majus*, *Cardus*, *Hydrastis Canadensis*. Earlier studies on homeopathic drugs like *Arnica Montana*, *Ruta graveolens*, *Hypericum*, *Ginseng*, *Aconite* has shown to provide radio-protection, mainly against sub-lethal X-irradiation in mice (Khuda-Bakhsh, 1995). Recent work of Pathak et al (2003) have shown the traditional homeopathic drug *Ruta* ^ derived from *Ruta graveolens* was found to be effective in inducing cell death in human (HL 60 and MGR 1glicoma) and murine (K 1735 clone X 21) cancer cells and provide chemo-protection for normal human PBLs and B-lymphoid cells. Great advances have been made in the treatment of some forms of cancer and new advances in surgery, radiotherapy, and chemotherapy has led to an increase in cure rates, however, such interventions are often costly and beyond the reach of many cancer patients living in the developing world (Sikora, 1997). In the present investigation the major reasons the patient's sites for going and financial problems (Pal, 2002b). The majority of patients trying Psorinum therapy expressed satisfaction and felt emotionally stronger even though in many the medicines failed to produce any marked response. The present study indicates that 15 patients (5.4%) were irregular with the Psorinum therapy. This group of patient initially started the therapy but discontinued on the ground that they needed guaranty regarding their cancer cure. Raising false hope in vulnerable people can also be regarded as direct harm (Ernst, 2001). Hence, no patients participating in the Psorinum therapy were given any guaranty of cure. Many physicians find it surprising, but

patients are usually the best judges of what works for them (Coulehan, 1999). Like earlier studies (Esinberg et al., 1993) we found that patients with higher levels of education and poor health status are likely to be an alternative medicines user. In the present study 65% of the patients were highly educated and some of them have even explored the Internet for cancer information. The patients were fully aware and convinced regarding the potential harm and benefit of the alternative therapy they are exploring and have consulted their oncologists. In contrast to earlier studies conducted in western countries (Crocetti et al., 1998; Morris et al., 2000), the number of female patients trying this therapy was low. One of the many reasons for this difference was that this therapy was not offered to patients suffering from breast and cervical cancer. The incidence of cancer is rising rapidly in the developing world consequent to increased longevity due to control of infectious disease, tobacco abuse and environmental degradation. The biggest challenge before the clinicians now concerns the management of the rising incidence of cancer in developing countries, with little prospect of more resources becoming available to fight the disease (Mitra, 1999). In this scenario it is emphasized that general practitioners and paramedical staffs should play a greater role in cancer awareness and detection as well as management (Gangopadhyay and Mallik, 2003). It is estimated that above 50% cancers are curable if they are detected early and screening has got a role in early diagnosis.

Indian cancer patients face multiple problems that are unique and specific to this country (Pal and Mittal, 2004). At present, about one million cases of cancer are diagnosed each year in India. Majority of patients are diagnosed in advanced stages of malignancy and hence curative therapy is not possible in them. Many terminal patients with cancer try various natural and herbal medicines for treatment (Pal and Mittal, 2003). The present investigation indicated that the alternative cancer therapy Psorinum is very popular among cancer patients even though in many cases this therapy was ineffective. However, anecdotal reports (Chatterjee et al., 1995; Chatterjee et al., 1999; Chatterjee et al., 2004) indicate that this therapy may be helpful in improving quality of life in some terminally ill cancer patients. The therapy is well tolerated and incidence of toxicity is less because of individual dosing (Pal 2002c). The cost of the therapy was within the reach of many cancer patients belonging to the underprivileged segment of the society, contributing to its immense popularity in Kolkata. Whether this can be translated into a willingness to use similar natural compounds for cancer prevention and treatment purposes now needs to be analysed.

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We like to acknowledge the help and cooperation rendered by the oncologists, pathologists, surgeons, physicians, haematologists, nurses and social workers of many private nursing homes and government medical institutions of Kolkata viz. Calcutta Medical College, Shree Shukhlal Karnani Memorial Hospital (SSKMH), Chittaranjan National Cancer Hospital, Cancer Research Institute and Welfare Home, Thakurpukur, in the clinical management of cancer patients undergoing Psorinum therapy. In particular, we like to thank Prof. Subir

Kumar Dutta, Dept. Of Pathology, UCM, Kolkata; Prof R.M. Bramhacharya, Dept. Of Radiation Oncology SSKMH; Prof D. Das Gupta, of Tata Memorial Hospital, zMumbai, for their guidance. We thank Gautam Saha for helping us in data management and Ranjana Chatterjee for patients management. A part of this study was funded by late Dr. Rabindranath Chatterjee. The Research Associateship to SKP from ICMR, New Delhi is duly acknowledged.

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ABSTRACT BOOK



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ABSTRACT BOOK

TITLE

Non Conventional Cancer Treatment with Psorinum.

AUTHORS AND INSTITUTION

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ABSTRACT

Aggressiveness of Malignant Tumour and resistance to treatment, both Radiotherapy and Chemotherapy, are main hindrance to effective management of Cancer. New therapeutic medicines are being considered in these cases along-with or without primary treatment, are such drug is Psorinum a Homeopathic Medicine prepared from scrub, pus and slough of wounds caused by scabies. This Drug has been used on 298 cases, mostly advanced, not being benefited with usual form of treatment. This group of patients mainly comprised of Cancer of lung, G.I. tract, hepatobiliary tract, pancreas with few cases of head and neck and uterus. Complete disappearance of mass was noted in 52 cases, 45 patients died in this series as most of them were terminally ill with very low general condition. In most of the cases there was arrest of disease progression or improvement of general health, significant relief of pain, ascites and dyspnoea. The drug was administered orally on daily basis. NO untoward side effects were noticed. Median duration of survival for Cancer of lung, stomach, pancreas, gall-bladder, liver and rectum were 17, 22.5, 14.9, 13, 7.8 and 30.9 months respectively. Exact mechanism of action was not known, however the result indicates that the drug could be a non-specific immuno-modulator. Further research is needed for thorough evaluation of the potency and pharmacokinetics of the drug.

TITLE

Evaluation of depression in chemotherapy patients in kashan university of medical sciences - Iran 1998-99.

AUTHORS AND INSTITUTION

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ABSTRACT

In a prospective Study from June 1998 till Feb. 1999 in our Hematology - Oncology Clinic in Kashan - Iran 38 Patients Who were referred to medical oncologist, were assessed by Beck's test. Of 38 patients (M=19 , F=19) 43% had depression of which 56% , 33% , 11% had mild , moderate and severe depression , respectively. The Frequency of depression was lower in women (53% VS 42%) but women had more advanced degrees of depression and 25% of depressed women had severe depression . but there was no case of severe depression in men. The most frequent diseases associated with depression were CNS tumors (100%) GI Malignancies (67%) and hematologic malignancies (50%) also , depression was more frequent in illiterate people than the literate ones (65% vs 33%) . With regard to treatment modalities , The least degree of depression was in the group treated only with chemotherapy and 100% of patients treated with surgery , radiation therapy and chemotherapy were depressed.

TITLE

QUALITY OF LIFE ISSUES - INDIAN PERSPECTIVE

AUTHORS AND INSTITUTION

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ABSTRACT

Over the last decade health-related quality of life has been especially important in cancer patients because biomedical measures of the impairments are insufficient (the treatment is only a modifier of the course of the illness). Therefore the patients' subjective opinion as well as those of their family members should have greater weight in the assessment of treatment outcome. In this pilot study, a questionnaire devised by us was administered to patients and their family members. The questionnaire consisted of 2 parts, one part had 6 questions with 5 options each which had to be rated on a scale of 1 to 5 (1-most important & 5-least important). The other part (3 questions) asked their opinion regarding importance of quality of life (QoL). An equal number (70%) of patients & their family felt QoL should be considered in all patients irrespective of the type of treatment that they receive. More than 50% of patients & their family felt that the diagnosis of cancer should be disclosed to the patient. More than 50% of both groups felt that QoL in treatment decisions should be considered by the doctor after discussion with the patient. Overwork/lack of time by the doctor was rated as the most important factor preventing better interaction with the doctor. Fatigue was rated as most important while considering physical functioning. Feeling of depression & thinking about illness was scored almost equally by both groups while evaluating psychological aspects. Among social factors, insecurity in the presence of others scored the least. Wish to know more about sexual problems/implications was considered least important by both groups. Additional data will be presented.

TITLE

Inflammatory myofibroblastic tumour. A lesion often confused with malignancy.

Girish M. Moghe, Prasanna S. Joshi, Nirmala A. Jambhakar.
Tata Memorial Hospital, Mumbai, India.

ABSTRACT

Inflammatory myofibroblastic tumours (IMT) or inflammatory pseudotumours were first described in the lung and subsequently in many other organs of the body. The clinical, radiological and the gross features often suggest a malignant tumour. The histologic spectrum is wide and encompasses the plasma cell-rich lesions and the fibrous sclerosing types at the polar ends and a wide range of overlapping appearances in between. This leads to diagnostic difficulties. In this study, 15 IMTs have been analysed comprising five pulmonary and ten extrapulmonary lesions. There were nine men and six women between 3 to 66 years of age. The symptoms were minor and nonspecific in pulmonary lesions and were related to the mass lesion at the involved sites in extrapulmonary locations. The myofibroblastic nature of these lesions was not recognizable at the pre-operative diagnostic work-up. Surgical excision of the tumours was done in all 15 cases. Histology showed a mixture of myofibroblasts, inflammatory cells and vascularised stroma in varying proportions. The differential diagnosis included carcinomas and other soft tissue tumours depending on the site. Immunohistochemistry was of limited help. Fourteen patients responded favourably to surgical excision and one patient died of surgery-related complications. The purpose of this study is to highlight the diagnostic difficulties encountered in inflammatory myofibroblastic tumours.

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A CONVENTIONAL HOMEOPATHIC DRUG PSORINUM HAS BEEN UNCONVENTIONALLY EXPERIMENTED FOR CARCINOMA TREATMENT – EXPERIMENTAL OUTCOMES ON TWO PATIENTS

Asim Chatterjee

The New Resource, Kolkata

Conventional Homeopathic drug Psorinum has been unconventionally experimented on 54 different types of cancer patients. The results of these experiments have been evaluated in course of time. These experiments depended on the aid of some specific allopath physicians, chemists and established research centres. The application of these experimental methods by cancer specialists has shown surprising improvements on patients. Examples of two such patients may be cited. Unconventional application of Psorinum on cancer patients, specifically in case of Adino Carcinoma needs to be researched in depth by specialists. Cancer, in present world, is a vice to humanity but simultaneously is a great challenge to doctors as no solution or treatment has been found for the disease as yet. Surgery plays a vital role in treating cancer. It's necessary for mainly two reasons

- 1) To detect the disease and collect specimen of the affected portions of the body
- 2) To remove the infected portion of the body.

Even radiotherapy and chemotherapy help in cancer treatment. So, sometimes all surgery, RT and CT are together used or at times separately to both detect and treat cancer. Cancer at parts like gall bladder, respiratory system, tongue are treated by RT in order to reduce pain or restrict 'pathological fracture'. However other arrangements except radiotherapy may be effective in case of cancer at lungs or some other parts of the body. Chemotherapy is applied at the primary stage of Leukaemia, Lymphoma etc. Surgery and CT are simultaneously used for G.I. Tract and breast cancer. General health condition of the patient needs to be examined before applying CT. Medicines are given to a cancer patient so that different parts of his / her body and the cells remain effective. But attention should be paid to the reactions of the body. In specific cases, the condition of the patient is as important as the 'host factor' (parameter). Actually, application of chemotherapy, to a large extent depends on the patient's performance status, resistance power, nutrition, chemical reactions of the body, the constituents and functioning of blood and the result of the blood test to determine the effectiveness of liver. If the patient's performance status is below 50 of the Karnofsky scale then CT is not possible. In other words, CT is limited only when Karnofsky scale is above 50. In that case when the patient becomes weak or seriously ill neither CT nor RT is possible. Some temporary relief methods include medicines to reduce pain, supply of blood for haemoglobin, glucose and salt

water to balance level of water in the body.

In order to judge the effectiveness of homeopathy medicines in treating cancer, this research based decision needs to be mentioned. Examples of 54 cancer patients of performance status below 50 in the Karnofsky scale can be cited on whom CT and RT are not possible, as such along with conventional treatment, homeopathic drug Psorinum has been unconventionally but appropriately applied.

Ingredients and Methods of Application

The 54 cancer patients, on whom Psorinum has been applied, have been under conventional treatment since long. Indeed, they were under the supervision of experienced cancer specialists of renowned institutions. Modern technical methods like Radio Imaging, USG, CT Scan, Endoscopy, Citology, Haematopathology have rightly detected their disease and they were then provided with antibiotic, aminodrip, fat soluble, blood as well saline. At a much later stage Psorinum has been simultaneously unconventionally provided with conventional methods in order to retain its effectiveness. Besides, some diabetic cancer patients have been provided with insulin also. But RT and CT have never been applied on any patient at any condition.

Condition of the patient at different stages are keenly observed and the information are collected in due order and preserved with the help of modern technology. Specialists' advices are considered to decide the medicines of the patient. When Psorinum is applied, the patient's condition is observed and experimented at every stage.

Results & Discussions

Generally patients of Karnofsky scale below 50 cannot be given CT and RT. However, except in some specific types of cancer, CT and RT are not applicable even at initial stage. Surgery is not possible in the advanced stage of cancer patients. Experiments have proved that when Psorinum is applied to those 54 patients of Karnofsky scale below 50, positive results could be obtained. This research paper contains the details of treatment of 2 such cancer patients – (To see the table at additional page). This research was disorganized at the initial stage. Thus the research was carried on to understand the unconventional use of homeopathic drug Psorinum on cancer patients at the dying stage. Those cancer patients, who were being rejected by the conventional treatment process, get some rays of hope by this research.

At the second level, the field of the research is being decided. Modern research field is limited to treat cancer of stomach, liver, gall bladder, pancreas and some lung cancer where surgery is not possible. Pharmacodynamics and Pharmacokinetics research of the medicine is carried on and the drug is experimented on animal bodies as well to examine its effectiveness. The constant endeavour and the devotion to work of all medical scientists and specialists will surely enlighten the way to cure cancer in future. Till now there have been no other researches on Psorinum for cancer treatment. That's why no evidence based proof material could be

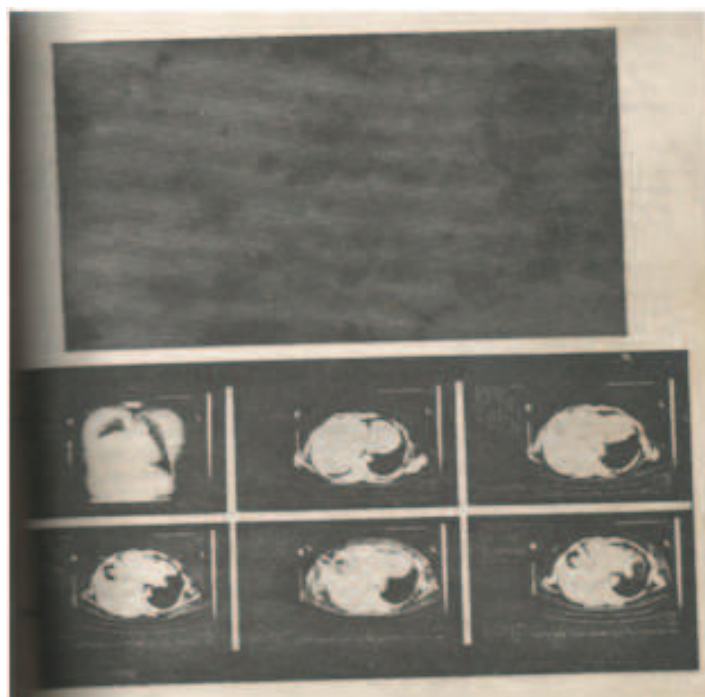
attached with this paper. In that way, this paper can be considered original and fundamental. Thus this paper is associated only with information based on the research.

I am grateful to several doctors who have helped me with their suggestions and advices. They are – Dr. RN. Brahmachari, Dr. Subir Kr. Dutta, Dr. R.S. Bhakta, Dr. Amiyakumar Hati, Dr. Prabir Kundu, Dr. Jharna Bhattacharya, Dr. Moloy Paramanik, Dr. I. Chatterjee, Dr. Hiranmoy Mukherjee, Dr. Pranab Bhattarcharya, Dr. D.C. Das, Dr. Partha Banerjee, Dr. Subrata Bhattacharya, Dr. Subhash Ghoshal, Dr. Ashokananda Konar, Dr. Samrat Bhattacharya, Dr. Satyapriya De Sarkar, Dr. Biswapati Mukhopadhyay and Dr. Utpala Chattopadhyay. Similarly I am highly obliged to several institutions of Kolkata for their liberal aid. They are – Chittaranjan Cancer Hospital, N.R.S. Medical College and Hospital, Association For the Cultivation of Science, School of Tropical Medicine, University College of Medicine, Medical College Hospital, National Medical College and Hospital and S. S.K.M. College and Hospital. Besides, the inspiration, encouragement, love and affection of several people around me have always been valuable for me – success is near with their expectations.

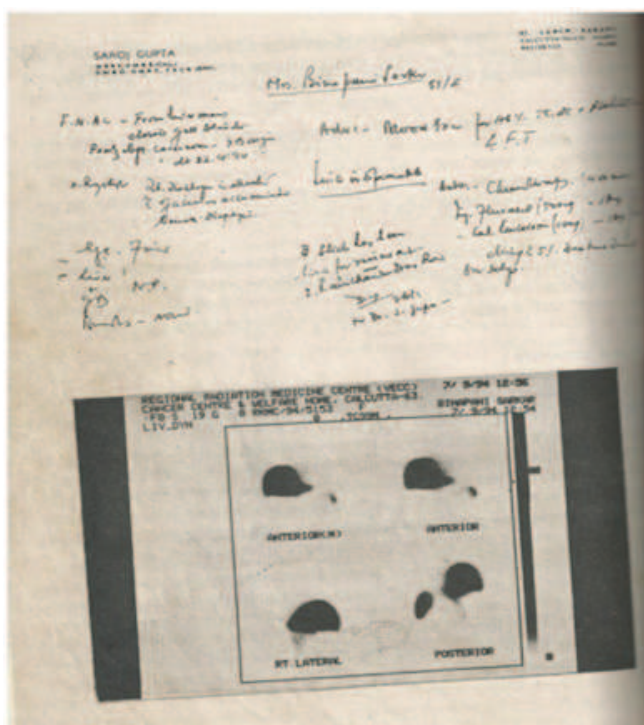
The doorway of highly technical and expensive treatment to cure cancer is almost bolted today. The whole world is trying hard to decipher ways for the quick recovery from this illness. Age-old treating procedures are prevalent in 3rd world countries like India, China, and Egypt. Today, it is relevant to judge those methods in terms of one or assimilation of terms of various treatment processes. I truly belief that mutual cooperation with intellect, thoughts, different views, going beyond the units of in-depth analysis of modern technology would surely help in this judgment.

Adjoined Paper (1)

BINAPANI SARKAR, 51 year old lady was admitted to Kolkata Medical College and Hospital on 25.03.1994 due to pain and swelling on the upper and right side of stomach. Medical tests could detect a tumour there. She scored 4 in the E.C.O.G. scale. Moreover, C.T. Scan showed modular and gall bladder. C.T. guided biopsy on 22.04.1994 of liver and gall bladder detected Adinocarcinoma. She was advised C.T. But patient did not agree to it. 05.05.1994 saw the beginning of Psorinum application on her. She was slowly relieved from pain. Ascitis and tumour – both began to dissolve. Patient's health showed signs of improvement. She scored 0 in the E.C.O.G. scale. Thereafter nuclear Scan of liver and gall bladder on 09.09.94 could no more detect any ascitis or tumour. Later her liver function test on 25.11.94 showed no inconsistency.



BIJOY KUMAR HUI – A 75 year old man. In 1985 this patient underwent gastrojejunostomy due to a sore in the stomach. But there were then no trace of cancer in his stomach. In 1992 he was admitted to a nursing home due to loss of appetite, vomiting, loss of weight and bleeding in stool. Then gastroscopy detected cancer in his stomach. (No. 1 biopsy slide no. 962/9 Dr. S.M. Ghosh). Biopsy report mentioned about adinocarcinoma. C.T. Scan located indications of cancer in his liver. Patient was admitted to Chittaranjan Cancer Hospital (Reg. No. s/92/1626) C.T. was thought of but patient's physical weakness did not permit. As such homeopathy treatment began with unconventional application of Psorinum. From 1992 April, this treatment was stated. Gastroscopy in SSKM in the year 1993 showed a much reduced sore in stomach. Again biopsy was done which still showed the presence of Carcinoma. (Slide no. 1/3/92/s/553/93: Date 27.1.93).



From then onwards, homeopathy treatment continued. 1993 July – no presence of no sore in gastroscopy. Biopsy report also did not mention about cancer. (Slide no. S/4127/93). This person is still very well and continues normally with all his work.

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PSORINUM: A NON-CONVENTIONAL MEDICINE OF CANCER POSES A GREAT CHALLENGE TO THE PRESENT DAY SCIENTISTS OF INDIA

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The dreadful disease Cancer results in uncontrolled proliferation of multiple cells within human body that leads to internal disorder in the normal functioning of the body. Inspite of all out endeavor by the entire medical world coupled with ambitious research projects involving billions of Dollars throughout the world, the outcome is far from being satisfactory. Till date no concrete method evolved out for the eradication of the disease. Rather the limitations of the Conventional methods of treatment of Cancer are being exposed as the day progresses. In fact, the conventional methods of treatment of Cancer are being exposed as the day progresses. In fact, the Conventional method of treatment only provide certain partial measures like surgery, Radio therapy and Chemo therapy either independently or simultaneously with one another and invariably with limited success. Its application is more restricted when the patient becomes resistant to Radio therapy or Chemo therapy depending on their affected sites of the body and lower health status as well. These features inevitably promoted a significant number of patients all over the world to be drifted towards non-conventional medicines.

Though the method of treatment in case of these medicines are usually symptomatic in nature and largely depend on the clinical inspection of Doctor's eyes as it had been since the primitive days. These have little relation with Pathological findings and were applied on trial and error basis. But the advancement of medical Science may justifiably demand that diagnosis of disease must be based on pathological reports and radiographic finding and its treatment should also be conducted on a scientific basis of modern day the Pharmacokinetics and Pharmacodynamics and the molecular structure of the ingredients of the specific drug should be ascertained first. But in case of cancer things are quite different for a number of reasons.

Firstly in case of other disease the variety of the particular disease is limited within 2 to 3 types while in case of Cancer the variety is innumerable and its prognosis largely depends

on the exact site of the human body where the growth has taken place. Secondly Cancer is associated with the internal genetic disorder resulting into uncontrolled proliferation of multiple cells, while the other diseases arise either due to infection of bacteria or attack of virus from external sources or irreparable damage of limbs in any site of the body. So these two types of deficiencies of human body are required to be viewed from two distinctly different angles.

In case of disease other than Cancer the annihilation of the external agencies like bacteria and virus is the prime factor while in case of cancer, reconstruction or repair of the internal damages should be the main concern. Unfortunately, till date all the available conventional treatments try to restrict the proliferation of cells either by debulking the malignant growth by way of surgery or destroying the undesired cells by way of Radio therapy or Chemo therapy. Perhaps this inappropriate approach in solving the complex problem was the main hindrance to find out the proper solution. Since Cancer relates to internal disorder it calls for some steps which will generate a harmonious condition inside the human body in setting the things in order or to accelerate repair or healing up internal deficiencies in a considerable period of time the non-toxic nature of the medicine should be an essential precondition. In this given context non-conventional treatment of Cancer is gradually gaining momentum in spite of its so many shortcomings. Application of traditional indigenous medicines with lower cost and avoidance of modern sophisticated technologies sometimes give patients a false sense of relief and complacency. But without reference to pathological finding and escaping the great contributions which science has conferred upon humanity the entire exercise becomes wholly unscientific. In these cases where the diagnosis of the disease can only be made correctly by pathological reports how its regression or remedy can be ascertained without getting through these reports. This great incompatibility remains as a stumbling block in accepting a non-conventional medicine as a rational one. In the given perspective we however conducted our research programme on cancer during the last 25 years with a nonconventional Homeopathy medicine known as Psorinum keeping in view of the Socio-economic scenario of India.

The source of the drug Psorinum, a Homeopathic medicine is the alcoholic extract of the Scabies scrub, Slough and Pus cells and it was administered orally. Prior to this therapy all the diagnosed patients were evaluated and staged by a routine special diagnostic technique. A coordinated group of Pathologists, Oncologists and Radiologists was conducting this research work alongwith other experienced physicians in order to secure all types of palliative treatment necessary to prolong the life of the patients who are under treatment of our non-conventional medicine.

When the trial started the drug Psorinum was administered without any supportive care, so, though there was considerable shrinkage observed in case of solid Tumour but the mortality rate remain very high. Later when the supportive treatment viz. blood transfusion, pleural paracentesis, analgesic, bronchodilators and stenting and other palliative surgeries were tagged, the mortality rate declined considerably.

We tried to concentrate our attention in the affected sites of body mainly in Lung and GI tract where little scopes is left for the Conventional therapy and very good results were obtained. We have all through assessed the condition of the patients objectively and published it in different journals from time to time. Details has also been recorded in National and International Scientific Conferences. Till date remarkable results have been achieved by our cost effective non-toxic medicine which has proved that an anti-cancer agent is latent in this drug. But the Pharmacodynamics and Pharmacokinetics of this drug is yet to be ascertained and it poses a great challenge to rise to the occasion by the scientists of the future days.

Since this National Seminar on Biotechnology for Education & Research spells out in its objective to join the race towards achieving excellence in research and deriving its benefit for the betterment of the society, may we urge upon its chief exponents to extend their hand of cooperation to unveil the truth regarding molecular structure and modus operandi of this non conventional medicine so that the present day hypothesis of this unconventional medicine may be transformed into a full proof scientific formula and accepted to the whole world as the panacea for this dreadful disease.

**National Seminar on Biotechnology- Education and Research April 24th 2003, Dept. of Chemistry Regional Engineering college, Dugapur, Pg.21*

**Registration Certificate for New Horizon Centre for Cancer Research
and Treatment**

West Bengal Form No. 264.



পরিদর্শনের পর প্রাপ্তি বহন করে
কী মহ (নগদে) প্রদান করা
করে। আইনক: বাধ্যতামূলক।

Registration granted in
anticipation of the
information/facts stated
in the document being
correct and true.

Certificate of Registration of Societies

WEST BENGAL ACT XXVI of 1961

No. 94703 of 1999 -19 2000

I hereby certify that New Horizon Centre
for Cancer Research and Treatment,

has this day been registered under the West Bengal Societies
Registration Act, 1961.

Given under my hand at Calcutta
this fifteenth day of June
One thousand four hundred and ninety nine



ACTP-A 290-1992-93

Sanjib Choudhury
Registrar of Firms, Societies &
Non-Trading Corporations,
West Bengal.

NEW HORIZON CENTRE FOR CANCER RESEARCH AND TREATMENT

- President: Prof. Dilip Basu, Former Vice-Chancellor, Burdwan University, Burdwan
- Secretary: Prof. Anup Majumdar, Former Professor of Radiotherapy, Radiotherapy Dept. B. S. Medical College, Bankura
- Prof. Subir Kumar Dutta, Former Head of Dept. of Pathology, University College of Medicine (UCM)
- Prof. Radheshyam Bhakta, Retired Professor of Chest Medicine, Calcutta National Medical College
- Prof. Amiyo Kumar Hati, Former Director of Calcutta School of Tropical Medicine
- Dr. Amitava Ghosh Roy, FRCS, Surgeon, USA
- Dr. Asis Mukherjee, Former Consultant Hemato-Oncologist, MD, DNB, DCH, Fellowship in Oncology
- Dr. Soumitra Kumar Choudhury, M. Sc, Ph. D., Senior Scientific Officer-II, Chittaranjan National Cancer Institute
- Biswapati Mukherjee, Former Professor at S. N. Pradhan Centre for Neurosciences, University College of Medicine (UCM)
- Dr. Satyapriya De Sarkar, Former Consultant Gastro Surgeon, Dept. of Gastro Surgery, Nightingale Diagnostic Centre
- Dr. Animesh Dutta, MBBS (son of patient Milan Dutta)
- Dr. Bishnu Prasad Mukherjee, Former Lecturer at Dept. of Chemistry, R. E. College, Durgapur
- Prof. Chittaranjan Maity
- Mr. Kartick Chandra, Former Consultant Oil & Gas Fields Projects, ONGC, including Alternative Energy Resources Projects
- Dr. Barin Roy, Dental Surgeon
- Pushpendra Krishna Bhowmik, Retired from service
- Mr. Gobindo Das Ghosh, patient of CA lung
- Goutam Saha, renowned Social Worker

NEW HORIZON CENTRE FOR CANCER RESEARCH & TREATMENT



(Registration No. - S/947033 of 1999 - 2001)

381, S.K. DEB ROAD,
NUTAN PALLY, LAKE TOWN,
CALCUTTA - 700 048.
TEL : (33) 5344702
FAX : (33) ~~5345997~~ 5340085
EMAIL : nhcfcat@yahoo.co.uk


Dated: 4.11.2001

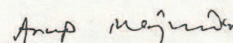
To,

Respected Sir,

The executive committee of 'New Horizon Centre for Cancer Research and Treatment.' will be highly honoured to have you with us as an honorary member in the advisory committee of the above mentioned society and request you to guide us in different ways with your enriched knowledge in research and management of Malignancy.

Thanking you.


Dilip Basu
President

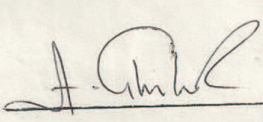

Anup Mazumdar
Secretary

**NEW HORIZON CENTRE FOR CANCER
RESEARCH AND TREATMENTS
381 S.K. DEB ROAD,
NUTAN PALLY, LAKE TOWN,
CALCUTTA - 700 048.**

(Registration No. - S/94703 of 1999-2000)

Form for Membership

1. Name in full (In Block Letters) : DR. AMITABHA GHOSH ROY
 2. Official Designation : _____
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 - Telephone No. : _____
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MILTON, MA., 02186, USA
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 5. Date of Birth : agroy@msn.com
 6. Personal Interest : _____
- Signature 

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Introduced By : _____

Membership No. : _____

Date 11 NOV-2001

Signature of
General Secretary : _____

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CALCUTTA - 700 048.

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- Telephone No. : 442 6555
5. Date of Birth : 9.4. ~~20~~ 1951
6. Personal Interest : MUSIC, READING CLASSICS
- : Signature Anup Mazumdar

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Date : _____

Signature of
General Secretary : _____

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CALCUTTA - 700 048.**

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(Registration No. - S/94703 of 1999-2000)

Form for Membership

1. Name in full (In Block Letters) : PROF DILIP BASU
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3. Office Address : RAJBATI, BURDWAN, 713 104
- Telephone No. : (0342) 62319
4. Residential Address : BLOCK-C, FLAT-4, PATIPUKUR GOVT. HOUSING
ESTATE 69/1 S.K. DEB ROAD, CAL-700048
- Telephone No. : (33) 534-7643
5. Date of Birth : 20.2.1940
6. Personal Interest : Social work, Science movement.
- : Signature Dr Basu

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Date : _____

Signature of
General Secretary : _____

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(Registration No. - S/94703 of 1999-2000)

Form for Membership

1. Name in full (In Block Letters) : DR. ASIS MDKHERJEE
2. Official Designation : Consultant Hemato-Oncologist MD, DNB, DeH.
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3. Office Address : 8/3 Alipore Rd. Calcutta-27
- Telephone No. : (33) 4567050-59 Residence - 4466362
Mobile - 98300-20196.
4. Residential Address : 51/1A Borojuri Mukherjee Rd
Behala Calcutta-24. 700034
- Telephone No. : (33) 4466362
5. Date of Birth : 10.4.67
6. Personal Interest : Leukemia and Bone Marrow Transplant

: Signature A. Mukherjee

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Introduced By : _____

Membership No. : _____

Date : _____

Signature of
General Secretary : _____

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NUTAN PALLY, LAKE TOWN,
CALCUTTA - 700 048.**

(Registration No. - S/94703 of 1999-2000)

Form for Membership

1. Name in full (In Block Letters) : Dr. SOUMITRA KUMAR CHOU DHURI, MSc., Ph.D
 2. Official Designation : Senior Scientific Officer, II
 3. Office Address : Chittaranjan Natl. Cancer Inst.,
37 S.P. Mukherjee Road, Cal-26
 - Telephone No. : _____
 4. Residential Address : CL 142/35; Salt Lake
Calcutta - 700 091
 - Telephone No. : 358 6267
 5. Date of Birth : 25.11.1954
 6. Personal Interest : Cancer Research & Welfare of Cancer patients.
- : Signature Soumitra Kumar Choudhury

(For Office Use Only)

Introduced By : _____

Membership No. : _____

Date : _____

Signature of
General Secretary : _____

LIST OF CANCER HOSPITALS (ALL STATES)

STATE	CITY/TOWN	CENTRE	UNIT
ANDHRA PRADESH	Guntur Pin 522 001	Govt General Hospital	
	Hyderabad Pin 500 004	MNJ Cancer Hospital & Radium Institute	Gammamatron ATC-9
	Hyderabad Pin 500 024	Bibi General Hospital	Gammamatron-R TH BOR
	Hyderabad Pin 500 029	Jagdish Cancer and Research Center	Phoenix
	Hyderabad Pin 500 001	Medwin Hospitals (Jaya Diagnostic)	Phoenix
	Kakinada Pin 533 008	Govt. General Hospital	Janus
	Kakinada Pin 533 04	Christian Cancer Centre	TH 780 C
	Kurnool Pin 518 001	Govt. General Hospital	TH 790 C
	Tirupati Pin 517 507	SVRR Hospital	TH 780 C
	Vishakhapatnam Pin 530 002	Kin George Hospital	EL 6
	Vishakhapatnam Pin 530 017	Vizag Hospital & Cancer Research Centre	Phoenix
	Warangal Pin 506 007	MGM Hospital	TH 780 C
ASSAM	Dibrugarh Pin 786 002	Assam Medical College Hospital	TH 780 C
	Guwahati Pin 781 002	Dr. B Barooah Cancer Institute Gammarex R	TH 280
BIHAR	Bokaro Steel City	Bokaro General Hospital	
	Jamshedpur Pin 831001	Tata Main Hospital	Philips XK 5100 Pilker –C
	Patna Pin 800 004	Patna Medical College Hospital	TH 60 TH 80

CHANDIGARH	Chandigarh Pin 160 0120	Post Graduate Institute of Medical Research	TH 780 C Varian
DELHI	New Delhi Pin 110 029	All India Institute of Medical Sciences	TH 60 CA
	New Delhi Pin 110 002	LNJPN Hospital Casea	Gamma Matron, TH 60
	New Delhi Pin 110 029	Safdarjung Hospital	EL 8, TH 780C Gammarex-R
	New Delhi Pin 110 029	Institute Rotary Cancer Hospital, AIIMS	TH 780, 780C Clinac 18
	New Delhi Pin 110 062	Batra Hospital and Research Centre	Alcyon II
GOA	Dona Paula Pin 403 004	GM Cancer Hospital and Research Instt.	Janus
GUJARAT	Ahmedabad Pin 380 016	Gujarat Cancer & Research Instt. (NP Shah Cancer TH 780 Hospital)	Atron, TH 60
	Baroda Pin 390 001	SSG Hospital	Gammatron R
	Jamnagar Pin 361 008	MP Shah Med. College & Irwin Gp of Hospitals	Gammarex R
	Rajkot Pin 360 008	Sh. NP Cancer Instt. Rajkot Cancer Society	Janus TH 780
	Surat Pin 395 001	Cancer Protection	Gammarex R
HARYANA	Rohtak Pin 124 001	Medical College Hospital	TH 765 TH 780 C
HIMACHAL PRADESH	Simla Pin 171 001	Indira Gandhi College Hospital	Gammarex R
JAMMU & KASHMIR	Jammu Pin 180 001	Govt. Medical College & Hospital	TH 780 C
	Srinagar Pin 190 001	Govt. Med. College & SHMS Hospital	Janus
	Srinagar Pin 190 001	Sher-I-Kashmir Instt. of Medical Sciences	TH 780 Mevatron 74
KARNATAKA	Bangalore Pin 560 002	Victoria Hospital	TH 60

	Bangalore Pin 560 029	Kidwai Memorial Instt. of Oncology	TH 780 C TH 60 Ceasa Gamma Gammarex-R Clinac 1800
	Bangalore Pin 560 004	The Bangalore Hospital	TH 780 C
	Gulbarga Pin 585 105	Peripheral Cancer Centre	Phoenix
	Hubli Pin 580 025	Karnataka Cancer Therapy & Research Institute	Gammarex R Janus, TH 780
	Mangalore Pin 575 001	TMA Pai Hospital & Research Centre	Phoenix
	Manipal Pin 570 119	Kasturba Cancer Hospital	Aleygn II TH 780 C
	Mysore Pin 570 016	Bharath Cancer Hospital	Aleygn II
KERALA	Calicut Pin 673 008	Medical College Hospital	Casea Gamma Gammarex R Gammatron R
	Ernakulam Pin 682 001	Govt. General Hospital	Phoenix
	Kottayam Pin 686 008	Medical College Hospital	Gammarex R
	Trichur Pin 680 553	Amala Cancer Hospital & Research Center	Gammarex R TH 780
	Trivandrum Pin 695 001	Regional Cancer Centre Medical College Campus	Gammarex R Janus, TH 780 Clinac 4
MADHYA PRADESH	Bhopal Pin 462 001	Gandhi Medical College & Hamidia Hospital	TH 780
	Gwalior Pin 474 001	Cancer Hospital & Research Institute	TH 780 TH 780 C
	Indore Pin 452 003	SG Cancer Hospital	EL 6
	Jabalpur Pin 482 003	Govt. Medical College & Cancer Hospital	EL 6
	Padhar Pin 460 005	Padhar Hospital	TH 780

	Raipur Pin 492 001	PT JNM College & Hospital	TH 780
MAHARASHTRA	Akola Pin 444 001	Sant Tukaram Hospital & Med. Research Centre	TH 804
	Aurangabad Pin 431 001	Medical College Hospital	Gammarex R
	Aurangabad Pin 431 210	Marathwada Cancer Hosp. & Research Centre	Alcyon II
	Barshi Pin 413 401	Nargis Dutt Memorial Cancer Hospital	Phoenix
	Mumbai Pin 400 026	Jaslok Hospital & Research Centre	TH 60
	Mumbai Pin 400 020	Bombay Hospital & Med. Research Centre	Janus
	Mumbai Pin 400 056	Dr. Nalabhai Nanavati Hosp. & Med. Res. Centre	TH 780
	Mumbai Pin 400 025	Lady Ratan Tata Medical Centre	
	Miraj Pin 440 003	Wanless Hospital Govt. Medical College	TH 60 Gammarex R
	Nagpur Pin 440 003	Rashtra Sant Tukdoji Cancer Hospital	TH 60
	Pune Pin 411 040	Malignant Disease Treatment Cen. Command Hospital	TH 80
	Pune Pin 411 001	Poona Medical Foundation Ruby Hall Clinic	TH 60
	Sangli Pin 416 416	General Hospital	TH 780 C
	Solapur Pin 413 003	Sri Siddheswar Cancer Hosp & Research Centre	Janus
MANIPUR	Imphal Pin 795 004	Regional Medical College	TH 780 C
MEGHALAYA	Shillong Pin 793 001	Civil Hospital	Gammarex R
ODISHA	Behrampur Pin 760 004	MKCG Medical College Hospital	Janus

	Burla (Sambalpur) Pin 786 017	VSS Medical College Hospital	Janus
	Cuttack Pin 753 007	AH Regional Centre for Cancer Research	TH 780
PONDICHERRY	Pondicherry Pin 605 006	JIPMER	TH 60
PUNJAB	Amritsar Pin 143 001	SGTB Hospital	EL 6
	Ludhiana Pin 141 008	CMC Hospital	TH 80
	Ludhiana Pin 141 009	LDU Cancer Hospital	TH 780 Mevatron 74
	Patiala Pin 147 001	GMC & Rajendra Hosp.	TH 760
RAJASTHAN	Bikaner Pin 334 001	SPMD & PBMG Hosp	TH 60
	Jaipur Pin 302 004	SMS Hospital	TH 780 C
	Jodhpur Pin 342 003	SNMC Hospital	Gammarex R
	Udaipur Pin 313 001	RNTMC & AG Hospital	Toshibarer 120
TAMIL NADU	Ambilikkai Pin 624 612	Christian Cancer Centre	TH 80 R
	Coimbatore Pin 641 037	Om Cancer Centre	Gammatron R75 Phoenix
	Kancheepuram Pin 631 502	Govt. Arinagar Anna Memorial Hospital	TH 780
	Chennai Pin 600 020	Cancer Institute Adyar	Casea Gammatron EL C ,TH 80R Clinac 4, 6/100 Picker C5 Philips
	Chennai Pin 600 004	Dr. KR Doraiswamy Memorial Cancer Centre	TH JR
	Chennai Pin 600 001	Govt. Hospital	Gammarex R

	Chennai Pin 600 003	Bernard Inst. Of Radiology & Oncology Govt. General Hospital	Casea Gammatron Gammatron 2 Janus
	Chennai Pin 600 018	Dr. Rai Memorial Cancer Institute	Gammatron 2
	Chennai Pin 600 008	Govt. Hospital for Women & Children, Egmore	Janus
	Chennai Pin 601 302	Govt. Royapettah Hospital Perumbakkam	Gammarex R Mitsubishi ACC
	Madurai Pin 625 020	Govt. Rajaji Hospital	Gammatron R
	Madurai Pin 625 009	Meenakshi Hospital & Reseach Centre	Mission Phoenix
	Neyyoor Pin 629 802	International Cancer Centre	Gammatron R TH 80
	Tiruchirapalli Pin 620 002	GVN Cancer Centre 16 Killedar St.	Phoenix
	Vellore Pin 632 004	CM College & Hosp.	EL 80, TH 60 Picker C9
TRIPURA	Agartala Pin 799 006	Cancer Hospital PO Kunjaban	Gammarex R
UTTAR PRADESH	Agra Pin 282 002	SN Medical College	EL 6
	Aligarh Pin 202 001	JN Medical College & Hospital, AMU	Casea Gammatron
	Allahabad Pin 211 002	KN Memorial Hospital	Janus TH 780 C
	Bareilly	Keshlata Cancer Hosp. Delpar	Phoenix
	Gorakhpur	Hanuman Prasad Poddar Cancer Hospital	Gammarex R TH 780 C
	Kanpur Pin 208 008	JK Cancer Institute	TH 80
	Lucknow Pin 226 003	RG Medical College	TH 780 C
	Lucknow Pin 226 001	Sanjay Gandhi Postgrad Inst. Of Medical Sciences	TH 780 C ML 20 MDY

	Varanasi Pin 221 002	Indian Railway Cancer Institute	TH 780
WEST BENGAL	Babkura Pin 722 102	BS Medical College Hosp.	Cesapan F
	Kolkata Pin 700 020	SSKM & PG Institute	Casea Gamma ATC C
	Kolkata Pin 700 020	Chittaranjan Institute	Picker 592 Gammatron ATC C/9
	Kolkata Pin 700 026	Chittaranjan National Cancer Research Centre	Picker 592
	Kolkata Pin 700 073	Medical college Hospital	Cesapan F TH B, TH 780 C
	Kolkata Pin 700 004	RG Kar Medical College	Cesapan F
	Kolkata Pin 700 004	NRS Medical College Hosp.	Cesapan F
	Kolkata Pin 700 063	Cancer Centre and Welfare Home	Gammarex R Picker C8

ABBREVIATIONS

Name	Full form
2 DXRT	Conventional external beam radiation therapy
3D-CRT	Three-dimensional conformal radiation therapy
5-FU	5-Flurouracil
6-MP	6-Mercaptopurine
8-OHdG	8-Hydroxydeoxyguanosine
A&E	Accident and Emergency
AACR	American Association for Cancer Research
ADA-SCID	Adenosine Deaminase Deficiency
ADH	Atypical Ductal Hyperplasia
AICR	American Institute for Cancer Research
AIDS	Acquired Immunodeficiency Syndrome
AJCC	American Joint Committee on Cancer
ALCL	Anaplastic Large Cell Lymphoma
ALH	Atypical Lobular Hyperplasia
AML	Acute Myeloid Leukemia or Acute Myelogenous Leukemia
ANC	Absolute Neutrophil Count
ANDA	Abbreviated New Drug Application
anti-TNF α	Antitumor Necrosis Factor Alpha
APC	Adenomatous Polyposis Coli
ara-CTP	Arabinofuranosylcytosine Triphosphate
ASCO	American Society for Clinical Oncology
ASCs	Adult Stem Cells
ATM	Ataxia Telangiectasia Mutated
ATP	Adenosine Triphosphate
ATRA	Atralin
AUSAID	Australian Agency for International Development
B cells	B Lymphocytes
BA/BE	Bioavailability and Bioequivalence
BCC	Basal Cell Carcinoma
BCS	Best Case Studies Programme
BCSH	British Committee for Standards in Haematology
BEACOPP	A kind of chemotherapy regimen

BM	Bone Marrow
BMI	Body Mass Index
BRCA1	Breast Cancer Type 1
BRCA2	Breast Cancer Type 2
C.T. Scan	Computed Tomography Scan
CA	Carcinoma
CA 125	Cancer antigen 125
CA 15-3	Cancer antigen 15-3
CA 27-29	Breast Carcinoma associated antigen or Cancer antigen 27-29
CAC	Circulating Angiogenic Cells
CAM	Complementary And Alternative Medicines
CARs	Chimeric Antigen Receptors
CAT-8015	Moxetumomab Pasudotox
CBA	Cerebral Attack
CBC	Complete Blood Count
c-crk	Cell Cycle Related Kinase
CD	Cluster Of Differentiation
CEA	Carcinoembryonic Antigen
CHART	Fractionated Radiotherapy Technique
CHO	Chinese Hamster Ovary
CMDA	Carboxy Peptidase
CML	Chronic Myelogenous Leukemias
CMO	Chief Medical Officer
CNCI	Chittaranjan National Cancer Institute
CNS	Central Nervous System
COPD	Chronic Obstructive Pulmonary Disease
CR	Complete Remission
CR	Complete Response
CRISPR	Clustered Regularly Interspaced Short Palindromic Repeats
CRT	Conformal Radiation Therapy
CSF	Cerebrospinal Fluid
CSF-1	Colony-Stimulating Factor 1
CT	Chemotherapy
CTx	Chemotherapy
CTX	Chemotherapy
D1	D1 Lymph Node

DCA	Deoxycholic Acid
DCIS	Ductal Carcinoma In Situ
DES	Diethylstilbestrol
DHFR	Dihydrofolate Reductase
DLBCL	Diffuse Large B-Cell Lymphoma
DMPA	Depot-Medroxyprogesterone Acetate
DNA	De-Oxy Ribo Nucleic Acid
DRDO	Defence Research Development Organization
DST	Directorate of Science and Technology
dUTP	2-Deoxyuridine-5-Triphosphate
EATCL	Enteropathy-Associated T-Cell Lymphomas
EATL	Enteropathy-Associated T-Cell Lymphomas
EBRT	External Beam Radiotherapy
EBV	Ebstein-Barr Virus
ECM	Extracellular Matrix
ECOG	Eastern Cooperative Oncology Group
EGF	Epidermal Growth Factor
EGF	Epidermal Growth Factor
EGFR	Epidermal Growth Factor Receptor
EGFR	Epidermal Growth Factor Receptor
EMeA	European Medicines Agency
EMT	Epithelial-Mesenchymal Transition
ENT	Ear, Nose And Throat
EPO	Erythropoietin
ER	Estrogen Receptors
ERCP	Endoscopic Retrograde Cholangiopancreatogram
ERT	Estrogen Replacement Therapy
ESCs	Embryonic Stem Cells
ET	Estrogen Therapy
FAK	Focal Adhesion Kinase
F-ara-ATP	Fludarabine Triphosphate
FBC	Full Blood Count
FDA	Food and Drug Administration, USA
FDG	F-18 Fluorodeoxyglucose
FdUMP	Fluorodeoxyuridine Monophosphate
FGF	Fibroblast Growth Factor

FL	Follicular Lymphoma
FLIPI	Follicular Lymphoma International Prognostic Index
FNAC	Fine-Needle Aspiration Cytology
FUTP	Fluorouracil Triphosphate
GAPs	Gtpase-Activating Proteins
GB	Gall Bladder
GBM	Glioblastoma Multiforme
G-CSF	Granulocyte-Colony Stimulating Factor
GCV	Ganciclovir
GCV-MP	Phosphorylating Ganciclovir to a Monophosphate Form
GCV-TP	Phosphorylation to the Triphosphate Form
GDP	Guanosine 5'-Diphosphate
GEFs	Guanine Nucleotide Exchange Factors
GFs	Growth Factors
GI	Gastrointestinal
GM-CSF	Granulocyte-Macrophage Colony-Stimulating Factor
GP	General Practitioners
GTP	Guanosine 5'-Triphosphate
G-U	Bladder, Penile, Vulva, Prostate
Gy	Radiation doses for cancer treatment are measured in a unit called a 'gray'
HAART	Highly Active Antiretroviral Therapy
HBV	Hepatitis B Virus
HCL	Hairy Cell Leukemia
HCV	Hepatitis C Virus
HD	Hodgkin Disease
HeLa	Henrietta Lacks
Her2	Transmembrane Tyrosine Kinase Receptor
HIF-1	Anthracyclines Inhibit Transcriptional Factor
HIV	Human Immunodeficiency Virus
HL	Hodgkin's Lymphoma
HLA	Human Leukocyte Antigen
HMRN	Haematological Malignancy Research Network
HNPCC	Hereditary Nonpolyposis Colorectal Cancer
HP	Histopathology
HPV	Human Papillomavirus

HRT	Hormone Replacement Therapy
HRT	Hormone Replacement Therapy
HSCs	Hematopoietic Stem Cells
HSCs	Haemopoietic Stem Cells
HSV	Herpes Simplex Virus
HSVtk	Herpes Simplex Virus Thymidine Kinase
HT	Hormone Therapy
HTLV-1	Human T-Cell Lymphotropic Virus Type 1
HTLV-1	Human T-Lymphotropic Virus
HUGO	Human Genome Organization
I.P.G.M.E.R.	Institute of Post-Graduate Medical Education and Research and Seth Sukhlal Karnani Memorial Hospital or P G Hospital (Post-Graduate) or SSKM Hospital
IAPM	Indian Association of Pathologists and Microbiologists
IARC	International Agency for Research on Cancer
IBC	Institutional Bio Safety Committee
ICCU	Intensive Cardiac Care Unit
ICMR	Indian Council of Medical Research
IFN	Interferon
IgA	Immunoglobulin A
IGF	Insulin Growth Factor
IGF-1	Insulin-Like Growth Factor-1
IGF1R	Insulin Growth Factor Type 1 Receptor
IGRT	Image Guided Radiation Therapy
IHBR	Intra Hepatic Biliary Radicles
IIT	Indian Institute Of Technology
IL	Interleukin
IL-2	Interleukin-2
IMA	Indian Medical Association
IMRT	Intensity-Modulated Radiation Therapy
INF α	Interferon-Alpha
INSA	Intelligence National Security Alliance
IORT	Intraoperative Radiation Therapy
IPI	International Prognostic Index
IRB	Institutional Review Board
ISV	Inferior Vena Cava

IUPAC	International Union Of Pure And Applied Chemistry
IV	Intravenous
JIMA	Journal of the Indian Medical Association
K-RAS	V-Ki-ras2 Kirsten rat sarcoma viral oncogene homolog
KSHV	Kaposi Sarcoma Herpes Virus
KV	Kilovolts
LCA	Lithocholic Acid
LCIS	Lobular Carcinoma In Situ
LD-50	Lethal Dose, 50% or Median Lethal Dose
LDH	Lactate Dehydrogenase
LDR	Low-Dose Rate
LET	Linear Energy Transfer
LHD	Left Hepatic Duct
lrECM	Laminin-Rich Extracellular Matrix
Lt.	Left
M. Sc.	Master of Science
M. Tech.	Master of Technology
MALT	Mucosa-Associated Lymphoid Tissue
MAPK	Mitogen Activated Kinase
MBBS	Bachelor of Medicine and Bachelor of Surgery
MCF-7	Michigan Cancer Foundation-7
MCL	Mantle Cell Lymphoma
MD	Doctor of Medicine
MDA-MB-231	Mitochondria of highly metastatic breast cancer cell line
MDCT	Multiple Detector Computed Tomography
MDR-1	Multiple Drug Resistance Type 1
MeIQ	2-Amino-3, 4-Dimethylimidazo [4, 5-F]Quinoline
MeIQx	2-Amino-3, 8-Dimethylimidazo [4,5-F]Quinoxaline
MEM	Minimal Essential Medium
MHC	Major Histocompatibility Complex
MHT	Menopausal Hormone Therapy
MIBG	Metaiodobenzylguanidine
MLA	Member of the Legislative Assembly
MOMP	Mechlorethamine, Oncovin, Methotrexate, Prednisone
MRCP	Magnetic Resonance Cholangiopancreatography
MRI	Magnetic Resonance Imaging

MSCs	Mesenchymal Stem Cells
mTOR	Mammalian Target Of Rapamycin
MUD	Matched Unrelated Donor
MV	Megavolts
MZL	Marginal Zone Lymphomas
MZL	Marginal Zone Lymphomas
NCI	National Cancer Institute, USA
NFAT	Nuclear Factor of Activated T cell
NGO	Non-Governmental Organization
NHB	Normal Human Brain
NHL	Non-Hodgkin Lymphoma
NHS	National Health Service, UK
NICE	National Institute for Health and Clinical Excellence
NIDR	National Institutes of Dental Research
NIH	National Institutes of Health
NK	Natural Killer
NLPHD	Nodular Lymphocyte Predominant Hodgkin Disease
NMRI	Nuclear Magnetic Resonance Imaging
NPM	Nucleophosmin
NSCLC	Non-Small Cell Lung Carcinoma
OCCAM O	Office of Cancer Complementary and Alternative Medicine
OCs	Oral Contraceptives
OT	Operation Theatre
PBSC	Peripheral Blood Stem Cells
PC3	Prostate Cancer Cell Line
PCA	Prostate Cancer Antigen
PCBs	Polychlorinated Biphenyls
PD-ECGF	Platelet-Derived Endothelial Cell Growth Factor
PDGF	Platelet Derived Growth Factor
P-E fever	Pel-Ebstein Fever
PET Scan	Positron Emission Tomography Scan
pH	A measure of the acidity or alkalinity of a fluid
PHARMA	Pharmaceutical Research and Manufacturers of America
PhIP	2-Amino-1-Methyl-6-Phenylimidazo [4,5-B]Pyridine
PHT	Post-Menopausal Hormone Therapy
PI3K	Hosphatidylinositol 3-Kinase

PICC	Peripherally Inserted Central Catheter
PR	Progesterone Receptors
PR	Partial Response
pRb	Retinoblastoma Protein
PSA	Prostate-Specific Antigen
PTEN	Phosphatase and Tensin Homolog
RAC	Recombinant DNA Advisory Committee
RAS	Reticular Activating System
Rb	Retinoblastoma
RBC	Red Blood Corpuscles
RCT	Randomised Controlled Trial
REAL	Revised European-American Lymphoma
REMoDL-B	Randomised Evaluation of Molecular Guided Therapy for Diffuse Large B-cell Lymphoma with Bortezomib
RGD	Recombinant Glycoprotein D
RHD	Right Hepatic Duct
RIC	Reduced-Intensity Conditioning
r-metHuSCF	Recombinant Methionyl Human Stem Cell Factor
RMO	Resident Medical Officer
RNA	Ribonucleic Acid
ROS	Reactive Oxygen Species
RT	Radiotherapy
Rt.	Right
RTKs	Receptor Tyrosine Kinases
RTx	Radiation Therapy
RV	Retroviral Vector
SCC	Squamous Cell Carcinoma
SCF	Stem Cell Factor
SCID	Severe Combined Immunodeficiency
SCT	Stem Cell Transplantation
SDF1	Stromal Cell-Derived Factor 1
SEER	Surveillance, Epidemiology, and End Result Program
SH2	Src Homology 2
SH3	Src Homology 3
SLE	Systemic Lupus Erythematosus
SLL	Small Lymphocytic Lymphoma

SNPs	Nucleotide Polymorphisms
SOL	Space-Occupying Lesion
SVC	Superior Vena Cava
T cells	T Lymphocytes
T-47D	Invasive Ductal Carcinoma
TAMs	Tumor-Associated Macrophages
TB	Tuberculosis
TBI	Total Body Irradiation
TGF	Tumour Growth Factor
TGFs	Transforming Growth Factors
THDF	Tetrahydrofolate
TKIs	Tyrosine Kinase Inhibitors
TKR	Tyrosine Kinase Receptors
TNF	Tumour Necrosis Factor
TNF- α	Tumor Necrosis Factor A
TNM	Tumor Metastasis Lymph Nodes
tPA	Tissue-Type Plasminogen Activator
TS	Thymidine Synthetase
TSGs	Tumour Suppressor Genes
UICC	Union for International Cancer Control
USFDA	United States Food and Drug Administration
USG	Ultrasonogram
UV Light	Ultra-Violet Light
VEGF	Vascular Endothelial Growth Factor
VEGF	Vascular Endothelial Growth Factor
VEGF	Vascular Endothelial Growth Factors
VEGFR	Vascular Endothelial Growth Factors
v-onc	Viral-Oncogene
VPCs	Retro Viral Vector Producer Cells
WBC	White Blood Corpuscles
WCRF	World Cancer Research Fund
WHO	World Health Organization
WM	Waldenstrom Macroglobulinemia or Lymphoplasmacytic Lymphoma
XRT	Radiation therapy
X-SCID	X-linked Severe Combined Immunodeficiency

PHOTO GALLERY

Centre to take care of terminally-ill

Dipankar Bose in Kolkata

Dec. 17. — For the first time in eastern India, a fully-equipped centre is being set up in a Salt Lake hospital for the treatment of terminally-ill patients, especially those suffering from cancer. The unit is named Terminal Care and Palliative Management Centre.

Final touches are being added to the centre coming up at Subodh Mitra Cancer Hospital and Research Centre, which is scheduled to be opened on Christmas morning.

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The new hospital unit will be inaugurated on 25 December. — The Statesman

in the next few months," the hospital secretary, Mr Ashok Bose, said.

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scope to be treated, the end is usually very painful. During the last days, these patients need proper help to do away with the pain. Since this terminal care centre will also cater symptomatic care, we believe that it would be capable enough to handle most of the needs of terminally-ill patients," a senior surgical oncologist, Dr Gautam Mukhopadhyay, said.

The hospital authorities have also promised that the non-commercial humanitarian hospice will offer beds at most affordable prices.

"Other than the prices on offer, special steps have also been taken to train the nurses, employed in this special ward. Moreover, the rules of the hospital would also be flexible for the patients of this terminal centre and their family members would be

allowed to spend extra hours than the usual visitors," said Dr Ashim Chatterjee, a renowned homeopath and one of the main brains behind the Centre.

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Aradeep Chatterjee

From: "White, Jeffrey (NIH/NCI)" <jeffreyw@mail.nih.gov>
To: "Aradeep Chatterjee" <aradeep_1@vsnl.net>
Sent: Thursday, November 03, 2005 3:09 AM
Subject: RE: Communication with Dr. White

Dear Aradeep,

While I write this I know that you are still traveling back home. I just wanted to take some time to say again how well I think you did at the meeting on Monday. I was very impressed by the way to quickly and thoroughly answered the questions from the committee members. I hope you feel good about how you did. You should be happy with yourself and your father and mother should be very proud of you. I will keep you informed of the progress towards a decision with regard to NCI's response to your case series.

Best regards,

Jeffrey D. White, M.D.
Director, Office of Cancer Complementary
and Alternative Medicine
National Cancer Institute, NIH
6116 Executive Blvd., Suite 609
Bethesda, MD 20892
phone: 301-435-7980
fax: 301-480-0075
e-mail: jeffreyw@mail.nih.gov
<http://cancer.gov/cam>

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Believe in the 'Best'

Twenty-one-year-old Aradeep Chatterjee, a homeopathic student of P.C.M. Homeopathic College, Calcutta, is the first medical student from India to have successfully performed the Best Case Series (BCS) programme.

The programme is conducted by the National Cancer Institute (NCI) which is under the National Institutes of Health in the US. The Best Case Series is meant to facilitate the diagnosis and treatment of cancer through alternative medicine.

Aradeep has now been invited to join the Advisory Panel Review of Cancer Therapy Evaluation Programme of the Drug Development Group to be held on October 31, 2005, in Maryland.

"I have to make a presentation on the research I have conducted based on the diagnosis of 25 cancer patients," he says. If Aradeep's presentation goes down well with the panel, the NCI will undertake a project to develop homeopathic cancer treatment through Psorinum therapy.

BERHAMPORE

Science and celebrations

On June 19, the Grant Hall of Berhampore played host to a science workshop to mark the centenary of Albert Einstein's Theory of Relativity and also to celebrate the International Year of Physics, 2005. The workshop was organised by Bigyan Bhavana and a large number of students from various schools and colleges in Berhampore took part.

The experiments held at the workshop familiarised students with the amazing world of science. An attempt was also made to make youngsters aware of how science can be used to perform tricks and 'magic'.

Then there was a quiz competition. The topic was obviously 'physics'. The function came to an end with some speeches on the life and works of Einstein.

Rizwana Kamal, Class X, Mary Immaculate School



DOCTOR AT LARGE:
Aradeep Chatterjee (right)

SILCHAR

Rotary fest

Recently, the Rotaract club of Assam University celebrated their charter presentation by organising 'Rotafest'. This was the first time in the history of the university that a fashion show was held. And that too with three unique rounds — Peace round, Be a Vegetarian round and Brides of India round.

As if all this was not enough, the out-of-this-world performance by India Shining, the new band from the Barak valley, set the audience on fire.

Ashim Bhattacharjee, fourth

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New drug hope for cancer patients

Orally Administered Medicine Shows Promise Against Six Types Of Cancer

Dyuti Banerjee/TNN

Kolkata: Diagnosed with stage-IV pancreatic cancer in 2001, Minu Dutta was in such an advanced stage of the disease that neither surgery nor chemotherapy was possible, and her expected survival was only about three months. As a last resort, she enrolled herself into a clinical trial of psorinum therapy. Complete tumour remission was observed within one year of treatment. After 10 years of diagnosis, she is still disease-free. "This is a new life for me," she gushes with relief.

Psorinum therapy is an active-immunotherapeutic anti-cancer treatment against six types of cancer — liver, gall bladder, stomach, pancreatic, lung and oesophagus. The key medicine is Psorinum 6x — a diluted alcoholic extract of pus found in scabies vesicles. The medicine is liquid in form and administered orally.

"According to the pre-clinical data, Psorinum 6x activates different immune-effector cells, for instance T-cells, and accessory cells like macrophages, dendritic cells and natu-

ral killer cells that can trigger a complex anti-tumour immune response," explains Dr Aradeep Chatterjee, who is also the research director of the Critical Cancer Management Research Centre, Kolkata.

Kashinath Saha's case was very similar to Minu's. In 2003, he learnt that he had stage-IV lung cancer and was told that his expected survival was only about five months. "I had given up on life. I couldn't afford the expensive chemo and everything. So I thought this is the end." He was one of the 95 lung cancer patients who participated in another of three clinical trials on Psorinum Therapy. "I am now cancer-free and leading a normal life," he added.

The cases of Dutta and Saha were reviewed and verified by the National Cancer Institute under the National Institutes of Health of USA as part of their Best Case Series Programme.

Psorinum therapy, however, has faced some skepticism among a few oncologists in the city. Dr Gautam Mukherjee says, "It is doubtful how far Psorinum can work independently without conventional lines of



treatment, like chemo or radiation therapies."

However, in the three clinical trials conducted till date to evaluate the efficacy of psorinum therapy, the participants' eligibility criteria included pathological "confirmation of malignancy, inoperable tumour, and no prior chemotherapy or radiation therapy". The details of all the three trials were reviewed by the American Society of Clinical Oncology (ASCO) and the outcomes published in premier peer-reviewed cancer journals including the Journal of Clinical Oncology (2009, 2010

and 2011). The first clinical trial involved 158 participants with advanced liver, gall bladder, pancreatic and stomach cancers, the second trial was conducted on 95 participants with lung cancer and the third trial was on 65 oesophageal cancer patients.

Dr Jaydip Biswas, director, Chittaranjan National Cancer Institute, Kolkata, commented: "The findings have been exciting and the journals support the clinical efficacy of Psorinum 6x, and confirm that it has no adverse side-effects." A section of experts, including Dr Amitava Chakraborty, assistant professor, department of radiation oncology at RG Kar Medical College, Kolkata, hail this novel treatment protocol as "a revolutionary work in cancer research".

University of Texas MD Anderson Cancer Center, Houston, USA, has signed a "mutual confidentiality agreement" on July 11, 2011 with Dr Aradeep Chatterjee to initiate follow up clinical trials on this novel cancer treatment protocol. Dr Siqing Fu, Assistant Professor at MD Anderson, recently corresponded,

"We are very excited to collaborate with Dr Chatterjee to bring psorinum 6x into allopathic cancer therapy." The therapy is already being replicated in Denmark.

Oncologist Dr Mikael Nordfors at Humlegaard International Cancer Centre, Copenhagen, Denmark, appears very convinced about the efficacy of this therapy: "Since I started with Psorinum Therapy, I've experienced a big improvement in results. I consider it to be perhaps the single most interesting treatment method for cancer that exists today," he said.

Each cycle of three months of the treatment costs only about Rs 3,500. A cancer patient needs approximately eight to 12 cycles of the treatment to complete a full course. According to Dr Aradeep Chatterjee, "Randomized, controlled clinical trials are now being planned to determine if psorinum therapy is better than the chemotherapies to treat those six types of cancer." If psorinum can live up to its promise, cancer patients around the world will have cause for relief, and cancer will lose its much dreaded edge, for sure.

'Cancer cure' draws world attention

Lake Town Medic's Claim To Alleviate Pain For Patients

By Arnab Ganguly
TIMES NEWS NETWORK

Kolkata: Cancer. The word is enough to strike terror in anybody's heart. Not for too long though, if a city-based homoeopath is to be believed. He claims in homoeopathy lies the future of research on and treatment of the killer disease. In fact, his own work on the subject has already earned worldwide recognition.

A visit by a senior official of United States' National Institute of Health to the obscure Lake Town address of Ashim Kumar Chatterjee next month will stand as a proof to the interest (if not anything else) the anti-cancer work has been able to generate.

On October 6 and 7, director of NIH National Cancer

Institute (Office of Complementary and Alternative Medicine) Jeffrey D. White will meet Chatterjee who has been working with terminally ill cancer patients over the past few years.

Of particular interest to the NIH official will be Chatterjee's papers published in International Congress of Oral Oncology, the second volume of Tobacco Counters Health, the International Association for the Study of Lung Cancer and an issue of the Lancet Oncology that refers

to Chatterjee's treatment.

Chatterjee was the only city-based doctor to present a paper at the World Assembly on Tobacco Counters

Health (WATCH) held in Delhi in March. White will also meet some of Chatterjee's patients after oncologists gave up hope and referred them to him.

"Some of the results are surprising. Many got symptomatic relief after trying out Chatterjee's treatment. We cannot comment on the scientific component

of the drugs he uses but it was effective in certain cases. Chatterjee is working in areas where modern medicine failed to work," said former head of radiotherapy department, Medical College and Hospital, Subir Ganguly.

"Chatterjee did not shun modern medicine and seeks technical support at times," said oncologist Gautam Mukhopadhyay.

Chatterjee has focussed treatment on lung, liver, pancreatic and gall bladder cancer and brain tumour.

He is willing to get his system tested by an expert committee and wants to amalgamate it with modern science. "The results are on the papers. Now it is for the people to decide whether to accept the treatment or not," he said.



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scope to be treated, the end is usually very painful. During the last days, these patients need proper help to do away with the pain. Since this terminal care centre will also cater symptomatic care, we believe that it would be capable enough to handle most of the needs of terminally-ill patients," a senior surgical oncologist, Dr Gautam Mukhopadhyay, said.

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That thing they do

The Americans have awards for everything, they really do. In which other country would you find an award for 'The Most Philosophical Kindergarten'? And like most other brain-related contests in the US, the winner was a desi. Five-year-old Shiruti Indiresan won her Socrates-successor prize with an essay where she wrote that happiness and beauty both came from truthfulness.

For the record

It can't be easy being Santa Mirza — if you're India's best-known (and richest) female sportsperson, everything you do, (eg, win one match at a prestigious tournament) makes history.



Santa Mirza

and every day is full of a billion expectations. In these times, it becomes important to set achievable goals in diverse fields. Take the example of international tennis glamour-girl Maria Sharapova, no stranger to pressure herself. No matter what else she achieves defending her title this year at Wimbledon, she can go home happy, knowing that she has topped her personal best grunting record with an impressive grunt of 101.2 decibels.

AWARD OF THE WEEK

Goes to Aradeep Chatterjee, a 21-year-old homoeopathy student from Calcutta, recently invited to join a National Cancer Institute (US) panel to speak on psoriasis therapy for homoeopathic cancer treatment.





Dr. Asim Chatterjee and Mr. Pushpen Bhowmik with two other foreign lung cancer specialists.

Dr. Asim Chatterjee with Prof. R.S. Bhakta at 2nd World Assembly on Tobacco Counters Health.



From left to right - Mr. Pushpen Bhowmik, Dr. Ranju Ralhan, Dr. Asim Chatterjee



Dr. Asim Chatterjee at 3rd World Assembly on Tobacco Counters Health



Dr. Asim Chatterjee with Dr. Gautam Mukherjee at 3rd World Assembly on Tobacco Counters Health



From right to left- Dr. Gautam Mukherjee, Dr. Asim Chatterjee, Dr. A. K. Varma



Dr. A. K. Varma and a renowned scientist with Dr. Aradeep Chatterjee



Dr. Aradeep Chatterjee at 5th World Assembly on Tobacco Counters Health



Dr. Aradeep Chatterjee with ex-union health minister Dr. Ramados and Dr. A. K. Verma



Left t right- Dr. Asim Chatterjee, Dr. Aradeep Chatterjee with an NRI oncologist



Dr. Goutam Mukhopadhyay



Dr. Subir Ganguly



Dr. Anindya Goswami with wife Dr. Swati Goswami and daughter



Dr. Asim Chatterjee with Dr. Anup Sadhu



Dr. Asim Chatterjee with Prof. Shisir Dutta



Dr. Hiranmay Mukherjee



Dr. Saraj Gupta



Prof. Jaydeep Biswas



Prof. Subir Dutta



Prof. Sudin Bhattacharya

My Mentors



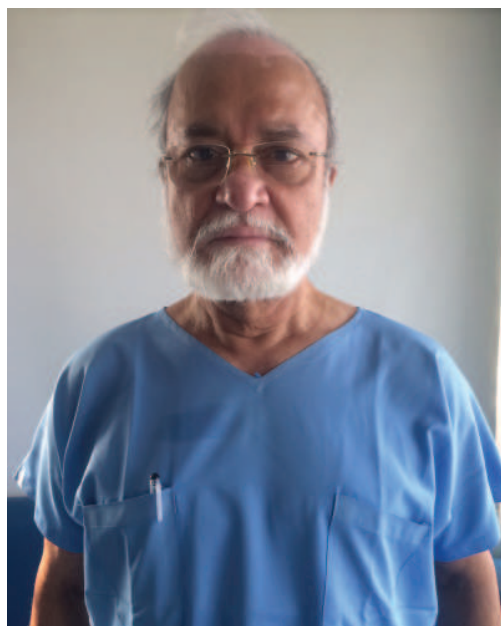
Prof. R.S. Bhakta



**Dr. Asim Chatterjee with
Prof. R. N. Brahmachari**



Prof. Anup Majumdar



Dr. Dipankar Dasgupta



Dr. Asim chatterjee with Prof. Jaydeep Biswas and patient Milan Dutta



Dr. Asim Chatterjee with Khoma Das (whose husband was a cancer patient) with daughter



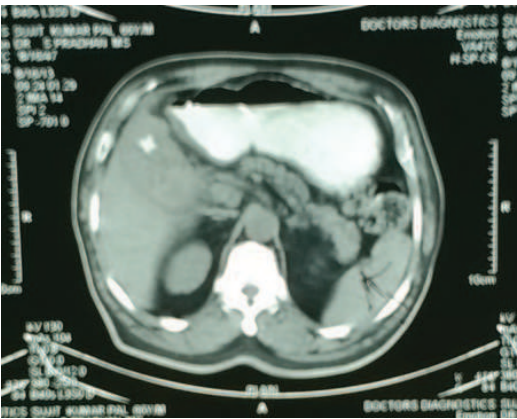
Dr. Asim Chatterjee
with Prof. Jaydeep
Biswas and Sujit
paul



Prof. Jaydeep Biswas and Sujit Paul



Sujit Pal



Sujit Pal 8.16.2013



Sujit Pal and his wife



Dr. Aradeep Chatterjee with Dr. Jeffery White



**Dr. Asim Chatterjee and Dr. Jaydeep Biswas
with Dr. Jeffery White**



**Dr. Jeffery White with Nandarani Banerjee
(patient at the left)**



Dr. Jeffery White with Sunil Karmakar (patient)



**Dr. Aradeep Chatterjee with Dr. Jeffery White
in America**



**Dr. Asim Chatterjee, Dr. Aradeep Chatterjee
and Dr. Jeffery White**



**Dr. Asim Chatterjee with a few renowned Cancer
Scientists**



Patient Kashinath Saha with Dr. Jeffery White



Left to right-Prof. Biswapati Mukherjee, Prof. R. S. Bhakta, Prof. B. P. Mukherjee, Prof. Anup Majumdar, Dr. Asim Chatterjee, Mr. Bhanja Choudhury, Kashinath Saha, Mr. Bhattacharya



New Resource Workers -Babua and Anjan

Left to right New Horizon members' meet Dr. Soumitra Choudhury, Dr. Satyapriya Dey Sarkar, Dr. Asim Chatterjee, Prof. Subir Dutta, Prof. Anup Majumdar and a lung cancer patient, Gobindo Das Ghosh



Left to right - Prof. Anup Majumdar, Dr. Hiranmay Mukherjee, patient Bimal Adhikary and Dr. Asim Chatterjee



Left to right - Pujan Babu, Mr. Sudeen Dasgupta (G.M. Subodh Mitra Cancer Hospital) and Dr. Asim Chatterjee



Left to right - Pro. Anup Majumdar, Dr. Hiranmay Mukherjee and patient Bimal Adhikary



Left to right - Dr. Asim Chatterjee, Smt. Binapani Sarkar, Prof (Dr.) Ghosh Roy, Dr. Satyapriya Dey Sarkar



Dr. Asim Chatterjee with Parul Bala Dey



Dr. Asim Chatterjee with Ganesh Maity (patient) and Pujan Babu



Dr. Asim Chatterjee with Ganesh Maity (patient)



Ganesh Maity (patient)



Babua and Anjan with Dr. Asim Chatterjee



Gobindo Mukherjee

My Family



Left to right-
Prof. Shisir Dutta,
Ranjana Chatterjee,
Dr. Asim Chatterjee,
Dr. Aradeep Chatterjee

Smt. Arati Banerjee
(mother-in-law of
Dr. Asim Chatterjee)



Smt. Ranjana Chatterjee
with daughter-in-law
Swarnali Chatterjee

Treatment of Oral, Lung, Liver, Gall Bladder, Pancreatic and Stomach Cancers Through Alternative Cancer Treatment Psorinum Therapy

Aradeep Chatterjee¹, Ashim Kumar Chatterjee², Joydip Biswas³, Sudin Bhattacharya³, Rita Chatterjee², Anup Sadhu⁴, Kalyan Kumar Mukherjee³, Durga prasad Nanda³, Bishnu Prasad Mukherjee⁵

1.A's Oncology Research & Treatment Institute, Kolkata, India 2.Subodh Mitra Cancer Hospital & Research Centre, Kolkata, India

3.Chittaranjan National Cancer Institute, Kolkata, India 4.Echo Diagnostic Centre, Medical College, Kolkata, India

5.National Institute of Technology, Durgapur, India

INTRODUCTION TO PSORINUM THERAPY

Why Complementary and Alternative Cancer Treatments are Gaining Momentum through out the World/ what are the weaknesses of the Conventional Cancer Treatments in general?

- Conventional cancer treatments are often too much expensive and beyond the reach of many cancer patients living in the developing world (Sikora 1997).
- Real treatment gains for the majority of cancers are still far from being satisfactory and the side effects of radiotherapy and chemotherapy have become increasingly intolerable to many (Cassileth et al 1996).
- In less developed countries majorities of cancer patients have less access to mainstream oncology and many cancer patients are never seen in hospitals (Sansom et al 2002).
- In most of the situations elderly cancer patients can't be provided conventional cancer treatment because of old age related problems (Turner et al. 1999).

What are the weaknesses of the Complementary and Alternative Cancer Treatments in general?

- Lack of proper scientific documents to prove the efficacy of Complementary and Alternative cancer Treatments (Chatterjee AK et al 2004).
- Treatment protocol is unfamiliar and complex that cannot be replicated easily in other clinical settings to get a sense of universality.
- Standardization technique of the medicinal power is unclear.
- Lack of Multidisciplinary team approach, Necessary infrastructure (Chaturvedi P et al 2002).

What is Psorinum Therapy?

- An Alternative Cancer Treatment that have the potential to cure or control all types of Cancer (Carcinoma, Sarcoma, Leukemia, Lymphoma).
- Very promising for the treatment of Oral, Lung, Liver, Gall Bladder, Pancreatic and Stomach Cancers.

What is the Background of Psorinum Therapy?

The concept of Psorinum Therapy was first proposed in the year 1980 by Ashim Kumar Chatterjee (Chatterjee A K et al 1995). Clinical Trial of Psorinum Therapy is incorporated into the clinical practice of CAM and other practitioners. This treatment protocol has been emerged out both from the ground of Homeopathy and Allopathy.

Is Psorinum Therapy a Homeopathy?

The treatment approach in Homeopathy is individualized and tailored to each patient. In Homeopathy the treatment protocol is Patient Specific Vs. Medicine Specific. In addition, Homeopathy believes in ultra molecular concept of Homeopathic remedies.

But, treatment protocol of Psorinum Therapy is Disease specific Vs. Medicine specific. In addition, anti-cancer medicine of Psorinum Therapy is based on the molecular concept instead of the concept of ultra molecule.

That is why Psorinum Therapy is not a Homeopathy.

Does the Psorinum Therapy have the basic weaknesses of Conventional and Complementary and Alternative Cancer Treatments?

- Psorinum Therapy is cost effective providing additional practical feasibility in the prevalent socio-economic scenario of the developing countries.
- The anti-cancer medicine of Psorinum Therapy is non-toxic which should be a great relief to the cancer patients.
- The anti-cancer medicine administration technique is easy as can be taken orally, the palliative treatment can be done most of the time

without living the patients' home. Therefore, hospitalization is less required.

- Older age cancer patients can easily adopt this therapy.

That is why Psorinum Therapy does not have the in general weaknesses of the Conventional Cancer Treatments.

- The evaluation of Psorinum Therapy is based on pathology, radiology and laboratory findings.
- Treatment protocol is familiar to the Conventional Cancer Treatments and can be easily replicated in other clinical centers to get a sense of Universality.
- Standardization technique of the medicinal power is clear.
- It has the multidisciplinary team approach, minimum necessary infrastructures, etc.

That's why Psorinum Therapy does not have the in general weaknesses of the Complementary and alternative Cancer Treatments.

PATIENTS AND METHODS

Settings:

This study was directed by the first two authors with the assistance of pathologists, radiologists, general allopathic physicians, otolaryngologists, chest specialists, gastroenterologists, nurses, other technical and non-technical persons and social workers. In recruiting the patients and to obtain other necessary suggestions, the guidance of oncologists was always ensured.

Patients:

The sample comprising 245 biopsy proved cancer patients (male 130 and female 115) of oral, lung, liver, gall bladder, pancreatic and stomach cancer (25 oral, 62 lung, 42 stomach, 40 gall bladder, 44 pancreatic and 32 of liver) aged between 22 to 85 years.

Inclusion and Exclusion criteria:

Only the patients of histological or cytological diagnosed oral, lung, liver, gall bladder, pancreatic and stomach cancer of both sex who had not received and not intended to receive chemotherapy, radiotherapy, surgery or any other anti-cancer therapy due to (1) financial constraints, (2) no treatment recommendation from the oncologists for poor prognosis and doubtful treatment outcome were selected.

The lower age limit was 18 years and there was no upper age limit for the eligibility.

Patients didn't able to understand written or spoken English, Hindi, or Bengali was not selected.

Written informed signed consent was taken from each patient before starting the study.

Staging:

- Staging has been done by TNM system.
- Patients' health performance status has been staged by Karnofsky Scale and by ECOG score.

Anti Cancer Medicine

- The anti-cancer medicine of Psorinum Therapy is Psorinum which is an alcoholic extract of the scabies, scrub, slough and pus cells.
- The drug Psorinum was administered orally at 0.01ml-0.02 ml/ Kg body weight/ day as a single dose on an empty stomach to all the patients. (Chatterjee AK et al 1999, 2002)

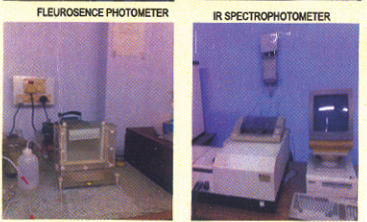
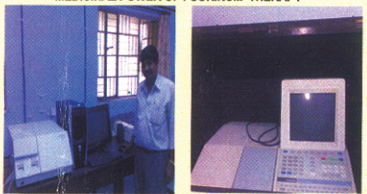
Supportive Treatment

The supportive care of this therapy to treat cancer associated ailments as well as other health related problems have been adopted from allopathic system. Supportive care for control of infection, pain, electrolyte balance, nutritional deficiencies, blood transfusion, abdominal or plural bleeding, paracetamol, analgesic, bronchodilator and stenting of the hepatopancreato-biliary system, bypass etc. were done as and when required (Chatterjee AK et al 2005).

Monitoring efficacy

- Diagnosis of cancer has been done by definite histological and cytological examinations.
- Comparative study of radiological follow-up has been done mainly by CT Scans and also by X-rays, endoscopy, ultrasounds etc. Proper radiological follow-up was done before the treatment began and after every 4 months interval and at the end of the study.
- Important and necessary lab reports have been meticulously collected and comparatively assessed.
- To assess quality of life, before the study begins and then after 3 months, 6 months, and at the end of the study, patients were asked questions about how they were doing during the treatment through interviews.

PHOTOGRAPHS OF INSTRUMENTS USED TO STANDARDIZE THE MEDICINAL POWER OF PSORINUM THERAPY



RESULTS

- Among the 245 cancer patients, 11 (4.49%) diagnosed at primary stage, 73 (29.8%) diagnosed at intermediate stage, 161 (65.71%) diagnosed at advanced stage.
- The therapy was offered mainly to those cases where the patients' Karnofsky Scale status was below 50% and ECOG Score status was 3-4.
- 200 (81.63%) have overall 1-Year survival, 156 (63.67%) have overall 2-Year survival, 140 (57.14%) have overall 3-year survival, 121 (49.39%) have overall 4-Year survival and 101 (41.22%) have overall 5-Year survival.
- Tumor response occurred in 132 (53.87%) cases among which tumor completely disappeared in 54 (22.04%) cases.
- After starting Psorinum Therapy, quality of life of the patients was improved almost in every case via Karnofsky Scale and ECOG score.

- Almost all the patients reported the therapy to be effective in reducing the cancer related pains, cough, dyspnea, nausea and vomiting, fatigue and improving appetite, liver function, etc.
- Patients who responded to this therapy also showed improvement in hemoglobin level, platelets, WBC counts, bilirubin, AFP level etc.
- A notable feature is the number of older age patients. Among the 245 cancer patients, the age of 136 (55.51%) were either 60 years or more and 76 (55.88%) of them and therefore, 31.02% of the total 245 cancer patients had died due to stroke and old age related problems.
- Patients report no side effect from the medicine of the Psorinum Therapy.

Relevant tables are illustrated as follows:

Table I

Primary Organ Affected	No. of Patients	Male	Female	TNM Staging of the Patients				Tumor Response Observed (%)	Tumor Completely Disappeared (%)
				Stage-I	Stage-II, Early Stage	Stage-III, Late Stage-II	Stage-IV		
Oral	25	14	11	2	11	12	72	72	05
Lung	62	32	30	2	23	37	48.39	19.35	14.28
Stomach	42	22	20	3	8	31	52.38	14.28	
G Bladder	40	21	19	2	11	27	80	17.5	
Pancreatic	44	24	20	1	8	35	47.72	18.18	
Liver	32	17	15	1	12	19	53.12	21.88	

Table II

Primary Organ Affected	No. of Patients	Male	Female	Overall Survival Data Analysis of the patients (N=245)				
				Overall 1-Year Survival	Overall 2-Year Survival	Overall 3-Year Survival	Overall 4-Year Survival	Overall 5-Year Survival
Oral	25	14	11	23	22	21	19	18 (72%)
Lung	62	32	30	51	35	32	25	21 (33.87%)
Stomach	42	22	20	34	24	21	20	18 (28.57%)
G Bladder	40	21	19	33	25	20	18	15 (37.5%)
Pancreatic	44	24	20	34	28	27	21	17 (28.64%)
Liver	32	17	15	26	22	19	17	14 (43.75%)

Relevant Figures are illustrated as follows:

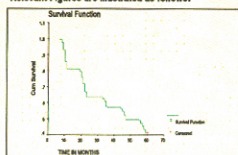


FIG. 1. OVERALL SURVIVAL PATTERN OF THE PATIENTS (USING KAPLAN-MEIER METHOD)

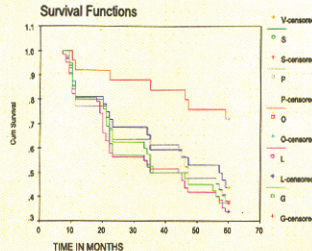


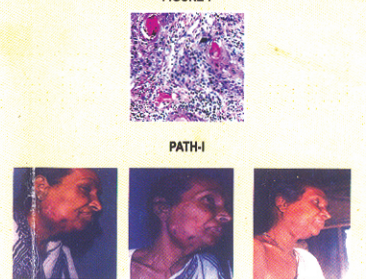
FIG. 2. OVERALL SURVIVAL PATTERN OF THE PATIENTS AS PER THEIR PRIMARY SITES (USING KAPLAN-MEIER METHOD)

V LIVER, S STOMACH, P PANCREAS, O ORAL, L LUNG, G GALL BLADDER

CASE: I

SBG 60 Year old female, a case of anaplastic squamous cell carcinoma of oral. She presented with T4 malignant tumor in the right mandible. Involvement of lymph nodes in both sides of the neck and more than 6 cm across. Metastasis in lung was present. She received only Psorinum Therapy and no other anti-cancer treatments.

FIGURE-I



CASE:III

PBD, 75-Year-old female, a case of squamous cell carcinoma of lung. She presented with T3 malignant tumor in the right upper lobe of the right lung. Involvement of lymph nodes is present. Metastasis in liver is present. She received only Psorinum Therapy and no other anti-cancer treatments.

FIGURE-II DESCRIPTION

PATH-I: Showing presence of gastric adenocarcinoma (Lauren- Intestinal type).

CT-I and CT-II: Showing presence of gastric tumor before Psorinum Therapy

CT-III and CT-IV: Showing regression of gastric malignant tumor 4 months after

Psorinum Therapy.

FIGURE:II DESCRIPTION

PATH-I: Showing presence of Squamous Cell Lung Carcinoma. X-RAY-I and X-RAY-II: Showing presence of T3 malignant tumor in the right lobe of the right lung. X-RAY-III and X-RAY-IV: Showing disappearance of the malignant tumor from the right lobe of the right lung 6 months after Psorinum Therapy.

CONCLUSION

- Interest in CAM therapies is growing rapidly and unless there is a dramatic improvement become lesser, CAM will continue to attract many cancer patients.

- Previously, interviews were conducted based on 300 biopsy proved cancer patients of Psorinum Therapy to know the patients and/or their caregiver's views about this therapy. The survey showed that the patients had tried this therapy mainly due to no treatment options, financial constraints, frustration with the conventional cancer treatments and believe in the efficacy of Psorinum Therapy and according to the survey, among the 300 cancer patients, 195 (65%) had consulted their oncologists before trying this therapy (Chatterjee AK et al 2005).

- Many oncologists, other health care providers, social workers, cancer patients and their caregivers now regard this therapy as very effective.
- Though, the exact mode of action of the drug Psorinum is still unknown, it is assumed that this alternative cancer therapy working as an immunomodulator by stimulating body's own defense mechanism.
- Patients report that the drug Psorinum is non-toxic and the therapy is also quite cost effective.

- This therapy can be easily replicated and translated by other practitioners in different clinical centers since:

- The reagent to prepare the anti-cancer medicine is available.
- The standardization technique of the medicinal strength is clear.

- The dosing of the medicine is clear (0.01ml-0.02 ml/ Kg body weight/ day) and the medicine administration technique is also very easy as can be taken orally. Patients need to take only a single dose in a day on an empty stomach.

- The supportive treatment has been taken from the conventional stream, which is the most familiar treatment protocol of the world.

- Although, this alternative cancer treatment have not been substantiated through case controlled study and the pharmacokinetics and pharmacodynamics of the drug Psorinum is still unknown, we should remember that:

(1) Among the 245, tumor response occurred in 132 (53.88%) among which tumor completely disappeared in 54 (22.04%) cases. The overall 5-Year survival was observed in 101 (41.22%).

(2) According to the TNM Staging the patients are mostly of intermediate (29.8%) and advanced stages (65.71%) and most of the cases, patients' Karnofsky scale status were below 50% and ECOG Score status were 3-4 with significant number of elderly cancer patients (55.51%) and 55.88% of them and therefore, 31.02% of the total 245 cancer patients had died due to stroke and old age related problems.

(3) All the patients received only the drug Psorinum with allopathic supportive treatment without having radiotherapy, chemotherapy, surgery or any other anti-cancer therapy.

(4) The cancer types are oral, lung, liver, gall bladder, stomach and pancreatic where chances of the spontaneous regression can be ignored considering the known evidences.

In addition, almost half of the total world's cancer burden is carried by developing countries that have access to very few of the resources available to fight the disease (Pat SK 2002).

Further investigations involving the pharmacokinetics and pharmacodynamics of the drug Psorinum and randomized controlled clinical trial should be done for further exploration of this therapy.

In the developing world and arguably in the developed world Psorinum Therapy may become an important component if integrated properly through proper scientific research into the mainstream oncology.

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